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Headache Sufferers' Psychological and Physiological Reactions to Stress.

J. Mark Pratt

Louisiana State University and Agricultural & Mechanical College

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HEADACHE SUFFERERS' PSYCHOLOGICAL AND
PHYSIOLOGICAL REACTIONS TO STRESS

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partical fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by

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December 1982

ACKNOWLEDGEMENTS

I gratefully acknowlegde the continued support, guidance, and encouragement that I have received during my doctoral training from my principal advisor, William Waters.

I am also indebted to Donald Williamson, Arthur Riopelle, Bernie Jensen, and Steve Bucu for their contributions as members of my dissertation and orals committee.

The present work was completed with the assistance of several colleagues and friends. Brian Bernard and Cathryn Wilkins provided experimental assistance and Helena Kramer was instrumental in helping me secure computer services at Stanford University. Special appreciation is also due to Rhea Cabin who prepared the manuscript.

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ABSTRACT

Relationships between stress and psychophysiological aspects of headache have often been assumed but seldom systematically demonstrated. The present study examined response patterns of 30 subjects, equally representing groups of migraine, muscle contraction headache (MCH) and low-frequency headache controls. Psychological variables assessed included the Beck Depression Inventory, a life stress scale, the Multiple Affective Adjective Checklist (MAACL), Levenson's Locus of Control Scales, and two subjective units of distress (SUDS) ratings of traffic fatality slides associated with emotional stress manipulations. Eight physiological measures (frontalis and trapezius EMG, temporal artery pulse volume, respiration frequency and amplitude, skin resistance level and conductance response, and heart rate) were monitored during a laboratory session. The 60-minute session consisted of three baselines followed by randomly presented conditions including relaxation, a mental test, and an emotional stressor involving the attempted induction of fear and anger.

Physiological findings were essentially five-fold. 1) Initial baseline differences among the three groups were negligible, except for trapezius EMG. However, Tukey's post-hoc tests did not identify differences between groups. 2) Significant main effects ($p < .05$) for stress condition were observed for six of eight physiological variables

(excluding skin resistance level and conductance response). Responses to fear and anger conditions were not differentiated. 3) Significant group differences emerged for respiration rate ($p < .05$). Tukey's test indicated that migraineurs' respiration rates increased during relaxation and decreased during emotional stress compared to controls who exhibited an opposite pattern. 4) A priori predictions of symptom-specificity were confirmed only for the migraine group in that during fear and anger conditions, migraineurs were significantly more dilated than MCH subjects ($p < .05$, $p < .05$, respectively). 5) Predictions related to migraine stereotypy were not supported.

Significant psychological differences ($p < .05$) among groups emerged for MAACL hostility, Levenson's internality, two ratings of laboratory stressors and life stress. Tukey's tests indicated that, compared to controls, MCH subjects reported more hostility, less internality, and more distress associated with the laboratory stressors. Migraine and MCH groups reported experiencing greater quality and quantity of life stress than did controls.

Finally, relationships among psychological and physiological measures did not appear significant. Implications of these findings for future research were discussed.

INTRODUCTION

Headache may be the most common medical complaint in the western world. Waters (1970) reports that 92% of his British Isles sample reported experiencing head pain at some point in their lives. The high incidence of this disorder has no doubt contributed to the extensive research addressed to the scientific understanding of this phenomenon over the last few decades. In spite of the pervasiveness of this disorder, a comprehensive classification scheme for headache was not proposed until 1962 when the Ad Hoc Committee on Headache proposed a list of 15 distinct headache types which are listed in Table 1.

Some headache types listed are clearly related to physical conditions such as trauma, infection, and metabolic or structural anomalies. Muscle-contraction, migraine, and mixed vascular and tension headaches, however, are typically classified as functional in nature (as opposed to organic), owing to the difficulty in targeting the physical contributions which may characterize these three headache types. These headaches without clear physical etiology account for 90% of the head pain complaints in clinical practice (Friedman, 1964). The prevalence of headaches of this type in the general population has been estimated at 80% for muscle-contraction (Ostfeld, 1962) and 5-10% for migraine (Kashawagi, McClure, & Wetzell, 1972). While migraine headaches occur most often among women between the ages of 20 and 45, children and males also experience migraines. Muscle-contraction headache (MCH) occurs equally often in both sexes and does not appear related to age.

Table 1

CLASSIFICATION OF HEADACHE
(Ad Hoc Committee, 1962)

1. Vascular Headache of Migraine Type
 - A. "Classic" Migraine
 - B. "Common" Migraine
 - C. "Cluster" Headache
 - D. "Hemiplegic" and "Ophthalmoplegic" Migraine
 - E. "Lower-Half" Headache
2. Muscle-Contraction Headache (MCH)
3. Combined Headache: Vascular and Muscle-Contraction
4. Headache of Nasal Vasomotor Reaction
5. Headache of Delusional, Conversion, or Hypochondriacal States
6. Nonmigrainous Vascular Headaches
7. Traction Headache
8. Headache Due to Overt Cranial Inflammation
- 9-13. Headache Due to Disease of Ocular, Aural, Nasal and Sinus
Dental, or Other Cranial or Neck Structures
14. Cranial Neuritides
15. Cranial Neuralgias

Headaches of the functional type often vary in their intensity and are frequently painful enough to interfere with personal functioning, causing the sufferer to seek professional help. Traditionally, the medical profession and drug industry provided the only treatment options open to headache patients. Recently, however, the combined efforts of some psychologists and physicians have supplemented available medical interventions with assessment and treatment techniques based on behavioral principles (Epstein, Katz, & Zlutnick, 1979). The result has been a global approach to the investigation of muscle contraction and migraine headaches which incorporates biological, environmental, and psychological considerations. Models illustrating this global approach include Bakal's (1975) "Biopsychological Perspective" and Diamond and Delassio's (1980) unified theory of migraine.

Distinguishing Characteristics of MCH and Migraine Headaches

Symptomatology

Migraine and MCH's are functional disorders which present very different clinical symptoms. The pain of MCH ranges from mild to severe, and typically is described as a feeling of pressure which extends around the head like a band (Lance, 1969). MCH pain is almost always bilateral and usually persists as steady pain ranging from two to six hours if not treated. This discomfort is associated with sustained contraction of the skeletal muscles of the head and neck. The muscular tension usually occurs as part of the individual's reaction to life stress (Ad Hoc Committee on Headache, 1962). Migraine headache typically involves severe pain that is often throbbing and unilateral

in nature. As seen in Table 1, vascular headaches of the migraine type are further subdivided into five categories, each associated with its distinct symptomatology. The term "migraine" designates any of these five subclassifications, and the origin of this name has been traced back to the first century, when Aretaeus of Cappadocia described the syndrome of unilateral head pain in detail and labeled it "hemicrania" (Diamond & Medina, 1980). Since then, the name has evolved to its present form.

Wolff (1963) described each of the migraine headache types in detail. The first, classic migraine, is distinguished by its sharply defined prodromal phase involving transient sensory-motor disturbances which may take the forms of blind spots in the visual field, flashing lights, vertigo, and numbness of the face and hands (Lance & Anthony, 1966). Nausea commonly accompanies the classic migraine headache. The onset of a classic migraine headache is usually unilateral and commonly occurs in the temporal, orbital, supra-orbital, or occipital areas of the head. The associated pain is usually reported as severe and often spreads from its point of origin during the headache episode to other regions. Pain usually lasts up to four to eight hours but may endure for several days. Common migraine headaches are very similar to classic migraines but occur without the prodromal phase and are less often unilateral than other migraine types. Cluster headache refers to the most excruciatingly painful of the migraine headaches, typically found in males and occurring in frequent episodes of short duration. These headaches tend to be seasonal and occur with little or no warning. Hemiplegic and ophthalmoplegic migraine are vascular headaches associated

with sensory and motor phenomena which persist during and after the headache. Finally, lower-half headache is thought to involve the vasculature of the lower face and occurs least frequently among vascular headaches.

Pathophysiology

Wolff (1963) is credited with providing the first comprehensive description of the mechanism characterizing MCH. His findings indicate that sustained contraction of the facial or neck muscles serves as an adequate stimulus to produce headache. Additionally, vasoconstriction of the vasculature providing blood and nutrients to the contracted muscles is associated with increased pain. Wolff reported that neck and facial muscle contraction often accompanies exposure to stress but that the pain may outlast exposure to the stressful stimulus.

The physiological concomitants of MCHs have been the frequent focus of laboratory studies. Sainsbury and Gibson (1954) were the first to document electromyogram (EMG) activity increases during headache. Similar findings have since been reported by Tunis and Wolff (1954), Friedman (1963), van Boxtan and van der Ven (1976), Malmo and Smith (1955), and Martin and Matthews (1979). Phillips (1977) however, found opposite results when measuring EMG activity during headache. In her study, recordings of four facial sites revealed lower EMG levels during headache compared to non-headache periods. Further obscuring the relationship between EMG recording and MCH is the failure of three studies (Bakal & Kaganov, 1977; Epstein, Abel, Collins, Parker, & Cinciripini, 1973; Harper & Steger, 1978) to find significant correlations between EMG levels and subjective pain ratings during

headache. More research in this area is necessary if we are to better understand MCH.

The sequence of events in migraine episodes appears to be more complex than is the mechanism described for MCH. Further, the pain associated with migraines appears to coincide with vascular changes rather than to muscular conditions as in tension headache (Wolff, 1963). The most frequent precipitating factor in migraine attacks is thought to be stress, but episodes do not usually commence until the stressor is removed. Diamond and Medina (1980) noted that the occurrence of migraine during post-stress periods is a hallmark of migraine. They also cited rapid hormonal changes as frequently involved in initiating migraine attacks, as evidenced in the elevated incidence of migraine during days preceding menstruation and during ovulation.

Diamond (1972) provides a detailed analysis of the migraine episode. The mechanism of the migraine attack is perhaps best characterized as a triphasic response to some environmental or endogenous trigger factor. The first phase of the migraine headache involves prolonged vasoconstriction of the internal and external branches of the carotid artery, resulting in limited blood flow, or ischemia, to the sites to which they normally provide nutrients. Restricted blood flow to the posterior cerebral artery, for example, is generally accepted as the underlying cause of visual disturbances specific to the preheadache phase in classic migraine. Other symptoms linked to this phase include speech difficulties, weakness, numbness, and dizziness.

The second phase in migraine headache is characterized by rebound vasodilation of the cephalic vasculature in response to brain and scalp tissue needs. This dilation is associated with cranial vessel expansion and the subsequent subjective experience of unilateral pulsing and severe pain. Other headache researchers have also implicated the roles of histamine and peptide kinins in the production of a sterile inflammation around the distended cephalic arteries (Diamond, 1972). The resulting edema and the maintenance of vessel pressure on free nerve endings is generally held responsible for the sensation of throbbing pain that characterizes migraine headache.

The final phase of the migraine attack, the post-headache period, involves the reduction of the local edema and the return of the vasculature to normal limits. Head pain diminishes during this phase but the affected head area may remain slightly swollen and tender for a day or more.

The physiological evidence supporting the triphasic vascular migraine mechanism has involved investigations of the hemodynamics of migraineurs before and during their headaches. O'Brien (1971b) documents vasoconstriction of both the internal and external carotid branches and found 20% mean reductions in blood flow to the brain and scalp during the prodromal phase of migraine headache. Other researchers (Skinhoj, 1973; Skinhoj & Paulson, 1969) have reported up to 50% reductions in intracranial blood flow during prodromes which are thought to account for the sensory-motor symptoms that accompany classic migraine attacks. The headache phase following arterial vasoconstriction involves a rebound dilation of blood vessels that is

associated with the pulsing or throbbing pain reports that characterize most migraine episodes (O'Brien, 1971a; Skinhoj, 1973; Tunis & Wolff, 1954). The headache phase also involves elevated blood levels of plasmakinin, histamine and serotonin, all of which are believed to lower pain thresholds and to produce the sterile inflammation and local edema common to vascular headaches (Fanchamps, 1974).

Biological Factors

The search for etiological factors in functional headaches has been the focus of much clinical inquiry. While it is true that exposure to stress appears to precipitate both muscle-contraction and migraine headaches in many cases (Bakal, 1975; Diamond, 1972; Diamond & Medina, 1980; Wolff, 1963), the incidence of migraine has been linked to numerous other factors which do not appear to be related to the onset of muscle contraction headache. For example, the incidence of migraine is greater among individuals with a positive family history for this disorder than it is for those whose parents do not suffer migraine (Dalessio, 1972). Refsum (1968) documented higher concordance rates for migraines between monozygotic twins than in dizygotic twins, but genetic contributions to MCHs have not been reported.

Certain foods appear to trigger migraine attacks but dietary contributions to MCHs have yet to be reported. Hanington and Harper (1968) note that diets containing cheese, sour cream, beans, and liver are high in tyramine content and may facilitate the release of norepinephrine, which acts as a general vasoconstrictor. Diamond and Dalessio (1980) report that other vasodilator substances such as alcohol, monosodium glutamate, and sodium nitrite, all common to the

American diet, are food for migraineurs to avoid. Elimination diets excluding foods with suspected vasoactive properties frequently are helpful in reducing migraine in selected cases (Lance, 1978).

Biochemical and pharmacological factors may also differentially effect migraine and MCH sufferers. Sicuteri (1972) has implicated serotonin as a major mediator of migraine headaches. Levels of serotonin have been found to drop precipitously in migraineurs just prior to and during the headache phase in 80% of subjects tested (Curran, Hentenberger, & Lance, 1965). Although the mechanism is not yet clear, the liberation of serotonin from blood platelets just before the migraine episode is thought to be responsible for the vasoconstriction and lowered pain thresholds that typify migraine headache (Sicuteri, 1973). Similarly, certain drugs are linked to the onset of migraine, but not MCHs. The use of estrogen in menopausal women or as a contraceptive has been reported to increase the probability of migraine by 10% (Carroll, 1971). Nitroglycerine, a powerful vasodilator, and reserpine, a serotonin antagonist have also been implicated as migraine triggers (Dasgaard-Nielson et al., 1974; Diamond, 1980).

Numerous attempts have been made to link physiological disorders to functional headaches, but these have been met with limited success (Adams, Feurenstein, & Fowler, 1980). Diastolic and systolic blood pressures were investigated in migraine sufferers by Selby and Lance (1960), but no differences between these individuals and controls were reported. Epilepsy in migraineurs and muscle contraction headache subjects have also been studied but Lance and Anthony (1966) reported no differences between these groups and controls. Lance and Anthony

(1966) also studied vascular, tension and no-headache groups for histories of allergies but did not establish significant findings.

Structural anomalies have also been more frequently implicated for migraineurs than for individuals with MCH. Clinically aberrant EEG records have been noted in migraineurs before, during, and between headache periods (Selby & Lance, 1960; Slevin, Faught, Hanna, & Lee, 1981; Strauss & Selinsky, 1945). Selby & Lance (1960) also reported that migraine sufferers more often reported histories of childhood vomiting and motion sickness than did other headache groups. Adams et al., (1980) reported this finding suggests the condition of "autonomic instability" which might characterize migraine sufferers. A recent study by Kuritzky, Toggia, & Thomas (1981) also revealed impaired vestibular functioning in migraine individuals. Lance (1978) cited research reporting pathological vascular histological studies in older migraineurs. This finding suggests migraineurs may be more at risk than the general population with regard to cerebral stroke.

Behavioral Treatments of MCH and Migraine Headaches

Until the last decade, drug therapies constituted the most common clinical treatments for migraines and MCHs. Analgesics, which raise the pain threshold, typically have been used with MCHs but have proven less successful with migraines (Diamond & Dalessio, 1978). Pharmaceutical treatment of migraine headache typically involves the prescribed use of vasoconstrictive agents such as propranolol, ergotamine tartrate, and methysergide maleate (Lance, 1978). The serious side effects associated with these drug therapies (tolerance,

nausea, insomnia, and potential damage to multiple organ systems) must be weighed against their efficacy estimates of 40 to 100% (Curran, Hintenburger, & Lance, 1967).

As was mentioned earlier, over the last ten years existing traditional medical treatments for headache have been supplemented by a number of behavioral interventions. Consistent with the clinical symptomatology of migraine and MCH, numerous studies have demonstrated that the instrumental or operant conditioning of certain physiological responses has been effective in reducing headache activity. The process of gaining control of autonomic physiological responses has become known as biofeedback, and evidence of its efficacy in treating migraine and MCHs has accumulated rapidly. A serendipitous observation at the Menninger Foundation served as the impetus for handwarming techniques in the treatment of migraine (Sargent, Green, & Walters, 1973). This occurred when a subject with migraine found that her sudden recovery from a migraine headache coincided with a ten-degree (F) rise in hand temperature. Since then, the handwarming technique has been frequently used in migraine research (Blanchard, Theobald, Williamson, Silver, & Brown, 1978; Johnson & Turin, 1975; Sargent, Walters, & Green, 1973; Silver, Blanchard, Williamson, Theobald, & Brown, 1979; Wickramasekera, 1973). The mechanism by which this procedure is thought to operate is the peripheral vasodilation and corresponding cephalic vasoconstriction that occurs with handwarming. This overall effect is also associated with a generalized decrease in sympathetic arousal (Benson, Klemchuk, & Graham, 1974; Sovak, Kunzel, Sternback, & Dalessio, 1978).

Migraine headaches have also responded to cephalic vasomotor feedback procedures. By reducing blood volume in the temporal artery a number of investigators (Allen & Mills, 1982; Feurestein & Adams, 1977; Friar & Beatty, 1976; Koppman, McDonald, & Kunzel, 1974) have reported success in controlling migraine headaches. The mechanism involved in cephalic blood flow control appears more directly related to the pathophysiology of migraines than does peripheral temperature control. Adams et al. (1980) suggested that cephalic vasomotor control mimics the actions of vasoconstrictive agents which are used to abort or prevent migraine headaches.

Specific biofeedback procedures have also been found to be effective in treating MCHs. Beginning with a study by Budzynski, Stoyva, & Adler (1970), numerous researchers have found that MCH individuals responded favorably to cephalic EMG biofeedback (Budzynski, Stoyva, Adler, & Mullaney, 1973; Chesney & Shelton, 1976; Epstein, Hersen, & Hemphill, 1974; Epstein & Abel, 1977; Haynes, Moseley, & McGowan, 1975; Phillips, 1975). Inasmuch as sustained muscle tension of the head and neck has been repeatedly implicated in the etiology of MCH, lowering cephalic muscle activity would appear to constitute a direct reversal of the MCH process.

Simple relaxation training has also been employed as a treatment for functional headaches. Studies which have examined the effects of teaching headache sufferers to relax have generally found this method superior to no treatment (Williamson, 1981). Blanchard et al. (1978) and Warner and Lance (1975) documented improvements in migraineurs who learned Jacobson's (1938) progressive relaxation procedure. Similar

improvements among MCH subjects using these techniques have been reported by Cox, Freundlich, & Meyer (1975), Haynes et al. (1975), Jacobson (1970), and Tatso and Hinkle (1973). Taken together, these studies suggest that reductions in levels of sympathetic arousal may account for much of the improvement ascribed to more specific biofeedback procedures. If this is in fact the case, the cost effectiveness of relaxation exercises over specific biofeedback procedures may indicate relaxation to be a superior choice in the treatment of headache. Controlled outcome studies comparing specific versus non-specific behavioral treatments of headache may serve to clarify this question.

Advances in cognitive behavioral therapy have also been applied to headache treatment. Mitchell and Mitchell (1971) combined elements of relaxation training and assertion training into a self-management package for migraine subjects and found that their combined program was more effective than relaxation exercises alone. Their initial success rate of 70% prompted them to expand their treatment to include other self-control techniques such as self-monitoring, thought-stopping, and desensitization. The expanded program was reported to have improved all of twelve migraine subjects (Mitchell & White, 1977). Significant though less dramatic improvements in MCH sufferers have since been attributed to cognitive coping skills training (Holroyd & Andrasik, 1980). Similarly, multi-modal cognitive-behavioral treatment plans emphasizing education, stress management, and self-control procedures including imagery, thought management, and relaxation have resulted in lowered headache activity in both migraine and MCH subjects (Bakal,

Demjen, & Kaganov, 1981; Cox, 1981).

The picture that emerges from a review of the literature relevant to behavioral approaches to the treatment of headache is complex. Considerable evidence exists suggesting that these various approaches represent viable alternatives to traditional medical treatments. In view of their safety and cost-effectiveness, behavioral interventions may often be preferable to traditional headache treatments.

Psychological Aspects of MCH and Migraine Headaches

As far back as 240 years ago, theories concerning the relationship between headaches and personality were formulated (Harrison, 1975). Wolff (1963) is credited with conceptualizing the "migraine personality" as manifesting traits of perfectionism, ambition, rigidity, and inhibited expression of anger. Friedman, von Storch, and Merritt (1954), in their observations of 2,000 tension headache patients, concluded that these individuals were typically anxious and suffered difficulties with emotional control. Unfortunately, the widely accepted notion that headache sufferers constituted clinical subgroups was not based on systematic scientific inquiry, but instead, was founded on interview data, dynamic interpretations, and unstandardized measurements (Adams et al., 1980; Bakal, 1975; Harrison, 1975). Other methodological flaws cited in the early headache literature include problems of diagnosis, the absence of multiple comparisons and controls, and selection bias (Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok, 1982). Although the term, "migraine personality" may describe a number of patients seen in clinical practice, Schnarch (1980) suggested that

clinical studies based on those headache patients who seek medical help may not be able to generalize their findings to the larger migraine population. Support for this position exists in Waters and O'Conner's (1975) finding that 46% of migraineurs who qualified for the diagnosis of migraine had never sought treatment. Bakal (1975) has concluded that the concept of the "migraine personality" has yielded to evidence demonstrating a great deal of idiosyncrasy and variability among headache sufferers.

More modern approaches to psychological assessment of headache populations have improved on the methodologies of earlier research. For example, considerable evidence has accumulated demonstrating significant differences between normals and headache sufferers on a variety of standardized self-report measures. Minnesota Multiphasic Personality Inventory (MMPI) profiles depicting slight elevations in sub-scales related to hypochondriasis, depression, and hysteria have been commonly reported for individuals with MCH and less often associated with migraine subjects (Harrison, 1975; Kudrow & Sutrus, 1979; Sternbach, Wolf, Murphy, & Atkinson, 1973). Similarities between headache sufferers' elevated MMPI profiles and those of other chronic medical patients, however, raise questions concerning whether the self-reported personality characteristics of distress are the cause or result of pain (Calsyn, Louks, & Freeman, 1976; Calsyn, Sperigler, & Freeman, 1977; Hanvik, 1951). Anderson & Franks (1981), in their study comparing MCH, migraine, and controls' responses on the Edwards Personality Inventory, reported MCH subjects appeared to be most insecure and anxious while migraineurs tended to score highest on measures of

rigidity, achievement orientation, and perfectionism. Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok (1982) measured four headache groups using seven instruments and replicated previous MMPI findings. They also found that headache subjects differed from controls on measures of psychosomatic discomfort and trait anxiety. They reported a pattern in which headache groups fell on a continuum, beginning with controls who demonstrated virtually no distress, continuing through migraine and combined migraine MCH groups, and ending with MCH subjects who reported the highest levels of distress. Similar patterns implicating MCH subjects as more highly distressed than migraineurs, and migraine subjects more distressed than controls have been reported (Pratt, Williamson, Cohen, Granberry, & Jarrel, Note 1).

Two additional self-report constructs which have only recently been examined in headache sufferers are individuals' perceptions of stress and their locus of control. Exposure to stressful situations has been generally accepted by health professionals and patients alike as a major contributing factor to headache onset. The definition of MCH by the Ad Hoc Committee on Headache (1962) implicitly links stress and prolonged muscular tension. Similarly, the onset of migraine headaches has been repeatedly associated with some form of stress (Dalsgaard-Nielson, 1965; Diamond & Dalessio, 1980; Henryk-Gutt, & Rees, 1973). Studies which have attempted to assess the impact of life stress on headache sufferers, however, have been few in number and inconsistent in their findings. Andrasik and Holroyd (1981) found no differences between various headache groups and controls utilizing

Holmes and Rahe's (1967) Social Re-Adjustment Rating Scale (SRRS). Pratt et al. (Note 1), however, reported the unexpected finding that control subjects endorsed more exposure to life stressors than did migraine, MCH, and mixed headache groups.

Assessing self-reports of stress from an exclusively quantitative approach may provide an incomplete picture concerning the extent to which individuals perceive their lives as stressful. Two alternatives to this problems which employ quantitative ratings to provide subjective evaluations of stress are Wolpe's (1973) use of Subjective Units of Distress (SUDS) and Saranson, Johnson, and Siegel's (1978) modification of the SRRS. This approach represents a situation-specific style of self-report that has been recommended by Mischel (1973). Use of this assessment method in headache research has only recently been attempted. Price and Blackwell (1980) compared the subjective responses of migraineurs and normals following their exposure to a film depicting a primitive ceremonial subcision rite. Migraineurs rated the film as significantly less distressing than did controls. Anderson and Franks (1981) found no difference between migraine, MCH, and control groups on the basis of their subjective ratings of a laboratory stressor. Pratt et al. (Note 1), employing a modified version of the SRRS, reported significant differences between migraine, MCH and control groups on their objective (SRRS scores) but not subjective (SUDS scores) ratings of life stresses. Questions concerning the role of stress perceptions among headache subjects persist and warrant further research in this area.

Perceptions related to expectancy constitute another concept that has generally been neglected in headache research. Rotter (1966) introduced the Locus of Control Scale to measure the extent to which individuals perceived events in their lives as controlled by themselves (internal control) or by luck, fate, and chance factors (external control). Subsequent modifications of Rotter's scale have resulted in a Health Locus of Control Scale (Wallston, Wallston, Kaplan, & Maides, 1976) and a Tri-Partitite Control Scale distinguishing between perceived control by internal, powerful others and chance factors (Levenson, 1973).

Subjects reporting internal orientations towards expectancy have been found to differ from their external counterparts in a number of health-related situations. Internals, for example, are more likely to use seat belts (Williams, 1972), participate in voluntary exercise programs (Sonstroen & Walker, 1973), and be informed about health maintenance (Wallston, Maides, & Wallston, 1976). Additionally, studies have demonstrated that internals are generally superior to externals in their ability to learn in biofeedback paradigms (Catchel, 1975; Johnson & Meyer, 1974; Wagner, Bourgeois, Levenson, & Denton, 1974). Kilmann, Laval, and Wanless (1978) found that internals were more likely to describe stressful life events in less distressing terms than did externals.

The investigation of self-report patterns among headache sufferers may clarify several issues in this area which are currently obscured. Do headache groups reliably demonstrate different styles of self-report on psychological measures related to distress? What

relationships, if any, exist between a subject's rating of distress and physiological variables? Additionally, the roles of psychological variables in identifying stress-related etiology and predicting response to treatment may deserve exploration.

Assessment of expectancy among headache groups has also been rarely attempted. Andrasik and Holroyd (1981) reported no differences between MCH subjects and controls on the Health Locus of Control Scale. Pratt et al. (Note 1), however, found MCH subjects to endorse perceptions of control by chance factors and powerful others significantly more often than did migraineurs and controls. In the only other study examining this dimension among headache subjects, Cox et al. (1975) reported a tendency for successful subjects in relaxation and EMG biofeedback conditions to describe themselves as more internal than their unsuccessful counterparts.

In summarizing the literature concerning the role of psychological factors in migraine and MCH, it is apparent that numerous questions still exist. First, do self-report patterns on measures of distress distinguish headache sufferers from each other and from controls? Secondly, in light of considerable speculation linking stress and headaches, what is the role of perceived stress in headache? Further research into questions of this type may serve to clarify issues related to the etiology of headache disorders and treatment selection.

Psychophysiological Considerations of MCH and Migraine Headaches

The physiological responses of headache subjects to various forms of physical and psychological stimulation has also received considerable

attention in recent years. Inasmuch as psychophysiological investigations of headache populations have been influenced by developments in general psychophysiological research, a review of the literature reporting recent advances in this area is offered.

Individual-Response Specificity

The notion that in some individuals certain organ systems serve as major participants in stress reactions was proposed by Cameron (1941). He speculated that while some people chronically responded to stress with blood pressure elevations, others reacted with gastrointestinal symptoms. Empirical support for this position was originally offered by Malmo and Shagass (1949) who found that psychiatric patients with cardiovascular and headache symptoms reacted to mildly painful stimulation with increased cardiac or EMG activity, respectively. Malmo, Shagass, and Davis (1950) proposed the term "symptom-specificity" to account for the tendency for certain medical populations to respond maximally to stress with their afflicted organ systems. Replications of this concept have been documented for patients with arthritis, hypertension, and gastric ulcers (Engel & Bickford, 1961; Moos & Engel, 1962; Wolf & Wolff, 1947).

The application of the symptom-specificity concept to normal populations has been reported by Lacey, Bateman, and van Lehn (1953) and Lacey and Lacey (1957). They reported that normal individuals demonstrated consistent patterns of reactivity to stress such that specific physiological systems reliably demonstrated highest levels of activation. Lacey and his coworkers reformulated the concept of symptom-specificity to apply to normals and introduced it as

"individual-response specificity." A closely related concept, "individual-response stereotypy," refers to the tendency for individuals to demonstrate patterns representing stable hierarchies of multiple physiological responses.

Further investigations into questions of individual response specificity and stereotypy have indicated that the degree to which these patterns are exhibited by individuals varies between subjects (Engel, 1960; Wenger, Clemens, Coleman, Cullen, & Engle, 1961). Schnore (1959) presented evidence demonstrating that at least five variables (heart rate, respiration rate, neck and forearm EMG, and blood pressure) contributed to patterns that remained stable across low and high arousal conditions. These studies, taken together, suggest that individuals might be classified either on the basis of how fixed or random their patterns appear or according to the extent to which their patterns are disrupted by varying amounts of stress. The relationship between these variables related to specificity and stereotypy and psychosomatic disorders represent interesting questions which have yet to be fully explored.

Lipowski (1968) has postulated that response specificity underlies all psychophysiological dysfunctions. In responding to stress, some individuals habitually respond with one organ system which is particularly reactive. Repeated stressful stimulation causes this system to become dysfunctional and vulnerable to collapse. The organ dysfunction resulting from heightened levels of reactivity to certain stimuli is similar to the "weak organ" cause of psychophysiological disorders proposed by Alexander, French, and Pollock (1968). For

obvious reasons, the applications of the concepts of individual-response specificity and stereotypy to MCH and migraine headaches are attractive. Such approaches predict that MCH and migraines may largely be due to dysfunctional or hyperresponsive cephalic musculature and vascular systems, respectively. Empirical support for this position, however, has been mixed.

Stimulus-Response Specificity

Another approach commonly employed in psychophysiological research involves examining patterns of responding that are specific to certain stimulus situations. The principle of stimulus-response specificity postulates that patterns of physiological responses will be similar in a given situation and that patterns may vary when the situation is different (Andreassi, 1980). Cannon (1936) documented similar patterns of sympathetic nervous system activation in response to "emergency situations" that were commonly elicited by exposing subjects to situations involving anger, fear, and pain. Similarly, Selye (1946) pioneered research demonstrating relationships between prolonged stress and increased adrenal cortex activity, thymus destruction, and ulcerations of the gastrointestinal tract. In the absence of more precise descriptions of emotions based on specific response patterns, Duffy (1962) proposed that emotions were merely epiphenomenon of various levels of physiological activation.

More recently, however, researchers in this area have begun to elaborate on the emotional arousal patterns reported by earlier psychophysiological investigators. Sokolov (1963) proposed a mode of stimulus-response specificity in this Theory of the Orienting Response

(OR). He found that individuals attending to non-threatening stimuli typically demonstrated vasoconstriction in their fingers, vasodilation in their heads, and cardiac acceleration. Sokolov termed the response to noxious stimulation the "defensive response" (DR) and noted that it was similar to the OR in every respect except for the occurrence of cephalic vasoconstriction. Major problems with Sokolov's conceptualization have stemmed from failures of subsequent researchers to replicate his findings (Lang, Rice, & Sternbach, 1972).

A position receiving considerably more support than Sokolov's has been proposed by Lacey (1959). Simply stated, the "intake-rejection hypothesis" suggests that passive attentional (intake of external stimuli) and more active cognitive tasks (rejection of external stimuli) are associated with different metabolic demands on the organism. Attentional processes place relatively limited metabolic demands as evidenced by cardiac deceleration. Tasks requiring mental effort or the mobilization of energy to respond to a noxious or threatening situation are associated with higher metabolic demands and thus involve cardiac acceleration. Lacey and his coworkers also noted that skin conductance responses directly covaried with heart rate during cognitive tasks and noxious stimulation but not during situations involving attention and empathic listening. The term "directional fractionation of response" has been applied to relationships between physiological responses that have been observed to differ in various situations (Andreassi, 1980). Replications of these findings have been reported by numerous investigators (Andreassi, 1969; Davis, 1957; Hare, 1972, 1973; Wenger & Cullen, 1958). These studies suggest that more precise demonstrations of

stimulus-response stereotypy than those anticipated by Duffy's (1962) emotional activation position may be possible.

A diverse assortment of situations have been employed in investigations of stimulus-response stereotypy. In some cases, attempts have been made to elicit reactions to specific emotions (Averill, 1969; Ax, 1953; Lazarus, Speisman, Mordkoff & Davison, 1962; Wolf & Wolff, 1947; and others), while other investigations recorded physiological responses to various physical and psychological stressors not necessarily linked to specific emotions (Davis, 1957; Engel, 1960; Lacey & Lacey, 1970; Malmö & Shagass, 1949; and others). Examples of non-specific stressors have included thermal stimulation, cold-pressor tests, mental tasks involving arithmetic or verbal problem-solving, and visual, auditory and painful stimulation. The most revealing emotional manipulations reported in the literature involve the experimental induction of fear and anger, and ingenious paradigms have evolved to elicit these reactions in laboratory subjects.

In one of the classic studies, Ax (1953) created an atmosphere of fear in subjects by leading them to believe that an equipment failure was likely to deliver electric shocks to them. Anger was elicited in this study by treating subjects in an abusive manner. Ax reported that fear responses typically yielded increases in skin conductance level, respiration, and muscle tension peaks, whereas anger raised diastolic blood pressure, generalized muscle tension and skin conductance, but lowered heart rate. Support for Ax's (1953) position has been reported by Funkenstein, King, & Drolette (1954) and by Schachter (1957). Generally, these studies have concluded that fear is

associated with an autonomic response pattern resembling epinephrine-like stimulation whereas anger responses resemble more the autonomic pattern associated with mixed epinephrine and norepinephrine reactions. Additional support for Ax's results has been provided by experiments employing imagined fear and anger conditions (Weerts & Roberts, 1976). The literature, however, has also frequently revealed failures to differentiate fear and anger physiologically (Greenfield & Sternbach, 1972).

Psychophysiological Response Patterning Among Headache Sufferers

Numerous attempts have been made to replicate Malmö & Shagass' (1949) finding concerning symptom-specificity in MCH subjects. Similarly, aberrant cephalic vascular responses have been the focus of several researchers seeking to confirm or discount symptom-specificity in migraine headaches. Answers to these questions have proved to be both elusive and challenging.

Individual-Response Specificity in Migraine and MCHs

Considerable confusion has stemmed from the lack of agreement reported by many headache investigators. For example, studies examining resting levels of cephalic muscular activity in individuals with MCH have been inconsistent in their findings. Evidence for higher EMG levels during rest for MCH subjects (Cohen, Williamson, Monguillot, Hutchinson, Gottlieb, & Waters, 1982; Martin & Mathews, 1979; Phillips, 1978; Pozniak-Patewicz, 1976; Vaughn, Pall, & Haynes, 1977) have been balanced by studies reporting no significant differences between MCH and control groups (Acosta, Jamamoto, & Wilcox, 1978; Anderson & Franks,

1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Bakal & Kaganov, 1977a; Gannon, Haynes, Safranek, & Hamilton, 1982; Tunis & Wolff, 1954; Sturgis, Note 2).

Clear distinctions between headache groups based on cephalic EMG activity have been further obscured by studies reporting higher cephalic muscle tension in migraineurs than in MCH individuals (Cohen et al., 1982; Phillips, 1978). Furthermore, Lader and Mathews (1971) have noted the relationship between subjective tension and cephalic EMG does not always appear to be direct. Blocking muscular activity with curare, they reported, does not necessarily lower subjective tension levels and reducing subjective tension with centrally-acting agents does not always reduce EMG activity. Clearly, definitive evidence for MCH symptom-specificity has yet to materialize.

Demonstrations of migraine-related individual-response specificity should, by definition, take the form of cephalic blood flow dysfunctions. It has been noted earlier that cranial vasodilation during and after headache have been related to the pathophysiology of this disorder (O'Brien, 1971a, 1971b; Skinhoj, 1973; Skinhoj & Paulson, 1969; Tunis & Wolff, 1953). Tunis and Wolff (1953) were the first to document that migraineurs, between headaches, typically exhibited greater temporal artery pulse wave magnitudes (cephalic vasodilation) than did controls. This contrast was even more pronounced during migraine headaches. In addition to increased cephalic reactivity, these authors suggested that migraine individuals also demonstrate greater cranial vascular variability in the one to three days prior to their headaches.

In a similar investigation comparing MCH individuals to normals, Tunis and Wolff (1954) found that unlike migraineurs, MCH subjects exhibited chronically lower cephalic pulse waves compared to controls and that these differences became exaggerated during their headaches. It has been suggested by Bakal (1975) that the etiology of both migraine and MCHs may lie in the predispositions of these populations to react maladaptively to neutral stimuli. Subsequent reports by Price and Tursky (1976) and Bakal and Kaganov (1977a) have provided some support for this position. Both studies found that individuals with migraine reacted to neutral situations with cephalic vasoconstriction as compared to the cranial vasodilation demonstrated by controls. Price and Tuskey (1976) also found MCH subjects to respond to similar non-threatening situations with cephalic vasoconstriction.

Some researchers have interpreted decreases in forehead temperature as cephalic vasoconstriction and decreases in finger temperature as evidence for cephalic vasodilation. Appenzeller, Davison, and Marshall (1963) reported that migraineurs reacted to a peripherally-applied thermal stimulus with digital vasoconstriction, not the vasodilation of the hands exhibited in normals. Although subsequent replications of this report were provided by Appenzeller (1969), Downey and Frewin (1972), and Elliot, Frewin and Downey (1973), several studies have reported no significant vascular response differences between migraineurs and controls (French, Lassers, & Desai, 1967; Hockaday, MacMillin, & Whitty, 1967).

The use of peripheral and cephalic temperatures as indices of cranial vascular activity continues to be common in headache research

(Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Cohen, McArthur, & Rickles, 1978; Cohen et al., 1982). Sovak, Kinzel, Sternback, and Dalessio (1978) reported that assumptions of cranial vascular activity from digital or cephalic temperature may not be entirely justified. Their findings indicated that skin temperature changes lag considerably behind vascular changes, sometimes by several minutes. In view of these findings, future headache investigators might benefit from assessing cranial vascular activity more directly than is possible with temperature recordings.

In summarizing the literature related to individual-response specificity among headache groups, it is apparent that questions in this area still present research challenges. The issue of MCH symptom-specificity is muddled by inconsistent relationships between subjective and objective tension levels (Lader & Mathews, 1971) and by the reports of some studies (Cohen et al., 1982; Phillips, 1978) that migraineurs demonstrated more cephalic EMG activation in response to stress than did MCH subjects. Similarly, the notion of migraine-related individual-response specificity has very limited support. Although several studies have linked the onset and course of migraine headaches to heightened cranial vascular reactivity and variability (O'Brien, 1973a, b; Skinhoj, 1973; Skinhoj & Paulson, 1969; Tunis & Wolff, 1953), few investigators have directly and systematically tested the effects of other stressors on migraineurs' cephalic vasculature responses. The failures of studies to demonstrate headache-related symptom-specificity and to reliably distinguish between MCH and migraine responses led Bakal and Kaganov (1977b) and Cohen (1978) to propose that migraine and MCHs were

not separate entities, but instead were similar disorders on a unitary continuum of pain intensity. As the next section illustrates, Bakal and Kaganov's (1977) and Cohen's (1978) position is still tenable.

Individual-Response Stereotypy in Migraine and MCHs

In the absence of direct evidence for MCH-related individual-response specificity, some researchers have focused their efforts on delineating more general patterns of physiological responses. A recent trend in headache research involves the investigation of patterns of physiological responses that distinguish headache groups from each other and from controls. Specific pattern differentiation for a particular headache group is generally accepted as evidence for headache response stereotypy, but results from studies of this type have been equivocal. Table 2 describes seven studies which have addressed the question of headache response stereotypy (Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Cohen et al., 1978; Cohen et al., 1982; Feuerstein, Bush, & Corbisiero, 1982; Gannon et al., 1982; Sturgis, Note 2). Methods of investigation in these studies typically involved comparing multiple physiological responses of headache subjects across various stress-related experimental conditions.

Results from this group of studies provide inconclusive evidence for headache response stereotypy. Cohen et al. (1978), Cohen et al. (1982), and Gannon et al. (1982) reported finding significant patterns of responding that differentiated migraine subjects from other groups. Additionally, Cohen et al. (1982) also documented evidence for MCH stereotypy. The lack of support for headache stereotypy cited by the remaining four (Anderson & Franks, 1981; Andrasik, Blanchard, Arena,

Saunders, & Barron, 1981; Feuerstein et al., 1982; Sturgis, Note 2) raises questions concerning the strength of positive findings in this area.

A major difficulty in comparing the inconsistent findings from these studies stems from the numerous methodological differences that characterize these investigations. While most studies cited compared migraine, MCH, and controls, two did not (Cohen et al. 1978 and Feuerstein et al. (1982). Sample sizes in these studies ranged from 26 (Cohen et al., 1978) to 196 (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982). Frontal EMG was the only dependent variable consistently employed by all studies. Measurements of cephalic blood flow have been less often reported. Experimental stressors in these studies varied in their number, duration, intensity, and type. All researchers, with the exception of Sturgis (Note 2) employed a mental test, usually a backward counting task or a general information test. Three studies utilized pain procedures including cold pressor tests (Andrasik et al., 1982), cuff pressure (Gannon et al., 1982), and digital pressure (Feuerstein et al., 1982). The use of emotional stressors was reported by Sturgis (Note 2) who exposed subjects to slides of mutilated human tissue and by Andrasik et al. (1982) and Feuerstein (1982) who engaged subjects in unpleasant imagery tasks. Most experimenters presented their multiple stressors in a fixed sequence, except Gannon et al. (1982) who employed a counterbalanced presentation of stressors to control for carry-over effects.

In view of the host of methodological differences that characterize this group of investigations, it is not altogether surprising that

Table 2

Summary of Seven Investigations of Response Patterning in Headache Groups

Authors	Headache Comparisons	Measures	Stress Tests	Results
Cohen, McArthur, & Rickles (1978)	MIG (13) Controls (13)	Frontalis EMG Forehead temp Hand temp Digital pulse volume Heart rate Skin conductance	Rest Orienting response Time estimation Reaction time Mental test Balloon bursting	Migraine response stereotypy (Migraineurs with more rigid response patterns across conditions)
Anderson & Franks (1981)	MIG (10) MCH (10) Controls (10)	Frontalis EMG Forearm EMG Hand temp Heart rate Blood pressure Heart rate Subjective ratings	Rest Step-up test Mental test	No evidence for headache response stereotypy
Sturgis (1981)	MIG (10) MCH (10) Controls (10)	Frontalis EMG Cephalic blood flow Digital blood flow	Rest Orienting response Aversive visual stimulation (Slides of mutilated human tissue)	Limited evidence for headache response stereotypy (Migraineurs with more variable cardiovascular responses) MCH subject with greatest EMG variability

Table 2 (continued)

Authors	Headache Comparisons	Measures	Stress Tests	Results
Cohen, Williamson, Monguillot, Hutchinson, Gottlieb, & Waters (1982)	Classic MIG (11) Common MIG (11) Mixed MIG-MCH (10) MCH (11) Controls (12)	Frontalis EMG Cephalic blood Hand temp Heart rate Skin conductance	Rest Orienting response Mental test Startle response	Headache response stereotypy (Headache group patterns were differentiated between each other and from controls)
Andrasik, Blanchard, Arena, Saunders, & Barron (1982)	MIG (39) MCH (62) Mixed MIG-MCH (37) Controls (58)	Frontalis EMG Forearm EMG Cephalic Blood Flow Hand Temp Heart Rate Skin Conductance	Rest Self-control tasks Mental Test Pleasant imagery Unpleasant imagery Cold pressor test	No evidence for headache stereotypy
Gannon, Haynes, Safranek, & Hamilton (1982)	MIG (16) MCH (13) Controls (15)	Frontal EMG Forearm EMG Earlobe blood flow Heart rate	Rest Mental test (and recovery) Cuff-pressure (and recovery)	Migraine response stereotypy (Migraineurs exhibited greatest degrees of vascular stress responsivity and rigid response patterning during recovery periods)
Fuererstein, Bush, & Corbisieros (1982)	MIG (11) MCH (9) Mixed MIG-MCH (11)	Frontalis EMG Neck EMG Cephalic blood flow Digital blood flow Skin conductance Heart rate Subjective ratings	Stressful imagery Mental test Finger pain	No evidence for headache response stereotypy

the patterns of responses offered as evidence for headache response stereotypy differ appreciably in the three studies reporting significant pattern differentiation among headache groups. Based on their findings, Cohen et al. (1978) rejected the hypothesis that migraineurs demonstrate generalized vasomotor instability. They described their migraine group as demonstrating more rigid patterns of response activation across stress conditions than did controls. Migraineurs, for example, demonstrated low levels of frontal EMG and higher head and hand temperatures across conditions relative to controls.

Cohen et al. (1982) partially replicated the Cohen et al. (1978) finding in that their classic and common migraine subjects had higher peripheral temperatures following relaxation. They also reported that their four headache groups demonstrated response patterns that were significantly differentiated from each other and from controls. Gannon et al. (1982) reported that migraine response stereotypy is especially evident during the recovery period following exposure to stress, and that this phenomenon is most evident in cardiovascular responses. Cohen et al. (1982) and Gannon et al. (1982), but not Cohen et al. (1978), interpreted their results as support for Wolff's (1963) position that migraine headaches are due, in part, to dysfunctional or hyperresponsive vascular systems.

In this group of seven studies, only Cohen et al. (1982) reported evidence for MCH symptom-specificity. These authors also noted that EMG responsiveness to stress was not differentiated between their migraine and MCH subjects. A closer examination of the few studies examining cephalic blood flow measures yields similarly inconclusive

support for migraine specificity, or stereotypy. Although Sturgis (Note 2) reported greater cephalic vascular variability during stress among the migraineurs as compared to controls, actual differences in responsivity between groups were not significant. Cohen et al. (1978) and Feurerstein et al. (1982) noted that all of their groups responded to noxious stimuli with cranial vasodilation, but no differences on this measure emerged between groups. One point of agreement shared by most investigators in this area (Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Cohen et al., 1982; Gannon et al., 1982; Sturgis, Note 2) is the prerequisite that stressors in headache research need to be sufficiently intense for specificity hypotheses to be amply tested.

Purpose of the Present Study

The preceding literature review suggests that numerous important questions still face headache researchers. The present study was designed to address the following questions.

1. Is there support for migraine and MCH symptom-specificity? Few researchers to date have succeeded in replicating studies demonstrating increased cephalic vascular responsivity in migraineurs and muscular responsivity among MCH sufferers (Malmo & Shagass, 1949; Malmo, Wallerstein, & Shagass, 1953; Tunis & Wolff, 1953, 1954). Support for hypotheses related to symptom-specificity of these disorders remains limited. It was hypothesized that if migraine- and MCH-specificity do, in fact exist, increased responsivity to stress in cephalic vascular and muscular measures for these groups, respectively, would be predicted.

2. Can migraine and MCH groups be differentiated on the basis of patterns of physiological responses? The large body of inconclusive results on this topic has prompted Bakal & Kaganov (1977) and Cohen et al. (1978) to postulate that migraine and MCH are really similar phenomena differing only in intensity of pain reports. Other investigators have suggested that migraine individuals have dysfunctional autonomic reactivity which may contribute to their disorder (Adams et al., 1980; Bakal, 1975; Cohen et al., 1982; Gannon et al., 1978). It was hypothesized that migraine-MCH differences do exist and that evidence for headache-related stereotypy should assume the form of differential response patterning among headache groups.

3. Assuming (1) that migraine and MCH specificity can be demonstrated, and (2) that response patterns specific to these headache groups can be demonstrated, under what circumstances are these conditions most likely to be observed? Most comparisons between headache groups and controls during conditions of rest and mild stress tests such as backward counting tasks have failed to produce headache classification-specific response patterns (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Cohen et al., 1982; and others). Most researchers in this area have agreed that intense stressors are necessary to adequately test hypotheses involving headache-related specificity and stereotypy. It was hypothesized that intense emotional stressors would provide greater responsivity and differentiation between headache groups than more commonly used mental tests.

4. What are the roles of specific emotional stressors in the physiological reactions of individuals with migraine or MCH? Investigations of specific emotions such as anger and fear have, in some cases, resulted in different physiological patterns (Ax, 1953; Funkenstein et al., 1954; Schachter, 1957; Weerts & Roberts, 1976). In view of considerable speculation concerning the role of emotional stress in headache etiology (Adams et al., 1973; Bakal, 1975; Harrison, 1975; Henryk-Gutt & Pees, 1973), the use of stressors specific to anger and fear represents a neglected area in headache research. It was hypothesized that physiological differences would be noted during fear and anger conditions for all subjects.

5. What differences exist between groups of headache sufferers and controls with respect to patterns of self-report on measures related to distress? Recent reports by Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok (1982) and Pratt et al. (Note 1) have indicated that headache groups may fall on a continuum of reported distress with MCH subjects reporting the highest levels and controls the lowest levels of distress. It was hypothesized that headache subjects would report more distress than controls and that MCH subjects would report higher distress levels than migraineurs.

6. What differences exist between groups of headache sufferers and controls on less-often employed self-report measures related to perceived stress and perceptions of control? The few studies which have investigated these psychological variables have provided inconclusive and sometimes inconsistent findings (Anderson & Franks, 1981; Andrasik & Holroyd, 1981; Price & Blackwell, 1980; Pratt et al., Note 1). No

specific hypothesis was proposed regarding perceived control differences between headache groups.

7. Finally, what relationships, if any, exist among physiological and psychological measures? Are physiological measures such as cephalic muscular and vascular responsivity and response stereotypy in any way predicted by psychological variables related to distress, control, and perceived stress? Studies examining these psychophysiological relationships among migraine and MCH sufferers have, to date, been nonexistent. No hypotheses concerning the relationships between psychological and physiological measures were proposed.

The present study represents an attempt to systematically examine some of the parameters commonly investigated in headache psychophysiological research. Previous researchers in this area have isolated numerous methodological shortcomings in headache studies and have proposed recommendations aimed at clarifying certain headache-related issues. Some of the improvements on previously reported headache research incorporated in the present study include: (1) the use of several self-report scales related to perceptions of distress and control; (2) the monitoring of multiple physiological responses including, among others, two cephalic muscle tension measures and cranial blood flow; (3) a comparison of the commonly employed mental tests to an emotional stressor involving fear and anger manipulations; and (4) counterbalancing of stress conditions.

METHOD

Subjects

Subjects were 30 individuals who responded to local media announcements inviting persons with and without headaches to participate in a psychophysiological assessment study. Initial contacts with respondents involved telephone interviews during which they received information about the experiment and then provided details for a screening questionnaire (see Appendix A).

The diagnostic criteria employed for the three experimental groups (migraine, MCH, and low-frequency headache controls) is presented in Table 3. Table 4 further describes characteristics of the sample. In addition to the Telephone Screening Questionnaire, a personal interview and a Headache Questionnaire (see Appendix B) were employed to facilitate accurate assignments of subjects to diagnostic groups.

Apparatus

Self-report data was collected using the following instruments: Headache Questionnaire (Appendix B), Life Change Scale (Appendix C), Beck Depression Inventory (Appendix D), Multiple Affective Adjective Checklist (MAACL) (Appendix E), Levenson's Tri-Partite Locus of Control Scale (Appendix F), Emotion Survey Form and SUDS Scales for visually presented stressors (Appendix G).

Subjects were seated in a comfortable reclining chair and were connected to a 12-channel Grass Model 7A polygraph with Beckman

Table 3
Sample Descriptions and Diagnostic Criteria
for Headache Groups

Group	Female/Male Ratio	Mean Age	Frequency	Diagnostic Criteria
Migraine Headache	9/1	29.3	> 1 per/mo	Three of following symptoms: 1) prodromal symptoms 2) unilateral focus 3) sharp throbbing pain 4) nausea and vomiting No use of vasoactive medications for 48 hours prior to participation
Muscle-Contraction Headache (MCH)	8/2	31.4	> 2 per/mo	No prodromal symptoms Dull, steady pain Bilateral focus
Low-Frequency Controls	8/2	28.6	< 3 per/yr	No indications of migraine or MCHs

Table 4
 Characteristics of Headache Subjects*

	Migraine n=10	MCH n=10	Controls n=10
Unilateral Pain	9	0	0
Bilateral Pain	7	10	3
Throbbing Pain	9	2	1
Steady Pain	3	10	3
Accompanying Nausea	6	2	1
Prodromal Symptoms	6	0	0
Headache Frequency			
< 1/mo	0	0	10
< 2/mo	1	0	10
< 3/mo	8	3	10
< 4/mo	8	7	10
< 5/mo	10	10	10
Medication Use**			
Mild Analgesics	4	7	3
Prescription Analgesics	8	6	1
Vasoconstrictors	3	0	0

*Information obtained from headache questionnaires (Appendices A and B) expressed in frequency of group endorsement.

**All subjects were evaluated in non-headache and non-medicated states.

silver-silver chloride electrodes. Specific recordings included: (1, 2) frontalis and trapezius muscle tension, recorded from electrodes at standard lead placements for frontalis and trapezius muscles (Phillips, 1978); raw EMG was recorded via Grass Model 7P511 a.c. amplifier/preamplifier and integrated through Grass Model 7P10 integrators and Grass Model 7DAF driver amplifier; (3) cephalic blood pulse volume, recorded by a photocystal reflectance plethysmograph securely held in place with a head sweatband against the left temporal branch of the external carotid artery and recorded via a Grass Model 7P122 d.c. amplifier/preamplifier; (4, 5) respiration frequency and amplitude, recorded by means of a strain gauge belt around the chest and a Grass Model 7P122 d.c. amplifier/preamplifier; (6, 7) skin resistance levels and responses, recorded with electrodes attached to the middle and forefinger of the left hand, and a 10 microamp current from the internal bridge of a Grass Model 7P122 d.c. amplifier/preamplifier; (8) heart rate, recorded from electrodes attached to left ankle and right subclavian artery sites, using a Grass Model 7P511 a.c. amplifier/preamplifier, a Grass Model 7P4 tachograph preamplifier and a Grass Model 7DAF driver amplifier.

Subjects remained seated in the sound-proof experimental chamber throughout the session and were in contact with the experimenter via a two-way intercom system. Verbal instructions and stress tests were presented via the intercom system. During the emotional stressor phases of the session, subjects viewed slides secured from the State Police Division of Traffic Safety and Driver Re-education depicting human traffic fatalities. Slides were projected with a Kodak Carousel

projector onto a 90 cm x 78 cm screen placed 148 cm directly in front of the seated subject.

Procedure

Individuals responding to media announcements were interviewed with the Telephone Screening Questionnaire. A general explanation of the assessment procedures was provided and respondents were informed that they might be contacted and scheduled for participation within 14 days. Following an evaluation of individual interview protocols, using the criteria outlined earlier, selected individuals were scheduled for laboratory sessions.

Upon arrival at the laboratory, a subject received a brief orientation and consent for participation in the study was obtained (see Appendix H). A subject was then provided with five self-report measures to complete (see Appendices B-F). Following this, the experimenter verified that the subject was not on potentially confounding medications and he/she was ushered into the experimental chamber and connected to the six recording channels of the polygraph. Once connected, the subject was instructed to sit quietly and to remain awake and to await further instructions. During this 10-minute adaptation period, individual polygraph channels were calibrated.

The experimental session involved exposing all subjects to each of three conditions--relaxation, a mental test, and emotional stress. Each of these three conditions was preceded by a 5-minute baseline. The order of the three conditions was randomized to control for carry-over effects (Gannon et al., 1982). Subjects were randomly assigned to one of six condition sequences: 1. Relax--mental test--emotional

stress; 2. relax--emotional stress--mental test; 3. mental test--relax--emotional stress; 4. mental test--emotional stress--relax; 5. emotional stress--relax--mental test; 6. emotional stress--mental test--relax. Physiological responses were continuously monitored throughout the session.

(1) The relaxation condition was introduced to subjects in the following manner:

For the next few minutes, please try to clear your thoughts and attempt to establish a deep state of relaxation. Avoid sleeping and try to move as little as possible.

At the conclusion of this 5-minute period subjects were told to relax and await further instructions.

(2) The mental test condition required subjects to respond to arithmetic and verbal problem-solving tasks. This stress condition, modeled after the "quiz electro-cardiogram" (Schiffren, Hartley, Schulman, & Ablemann, 1976) was introduced to subjects as follows:

For the next few minutes we would like you to solve some problems. Questions will be presented to you and after a short period a bell will sound. The bell is your signal to provide the correct answer. A perfect score on this test is indicative of genius ability but few people are expected to reach this level. Nevertheless, attempt to do your best.

Twenty questions comprised the mental test (see Appendix J) and each was followed by a 7-second period after which the signal sounded for subjects' responses. Following the first question, subsequent problems were introduced with the cue, "Next question . . ." At the conclusion of this 8-minute period, subjects were instructed to relax and await further instructions.

(3) The emotional stressor conditions took place over a 5-minute period. During this time, subjects were read the following script:

The slides you are about to view are on loan from the State Police Division of Traffic Safety and Driver Re-education. The scenes of traffic fatalities you will see are typically presented to impress on individuals with poor driving records the potential consequences of their actions.

This introduction was followed by further instructions for one of two conditions; the fear or the anger-provoking situation. These situations were presented randomly to control for sequence effects and were separated by a 45-second interval.

In the fear condition, subjects were read the following instructions:

As you view the following slides, imagine that you or your loved ones have been involved in accidents such as these. Reflect, if you will, on the personal impact of this event and the loss and suffering associated with it.

In the anger condition, the following script was used:

As you view the following slides, imagine that you were very close to the victim of this accident. Try to focus your reaction on the feelings you would have, not for the victim involved, but for the drunk and negligent driver of the other car who caused and survived the accident.

Subjects viewed two scenes of traffic fatalities for 30 seconds each in both the fear and anger situations. Following the instructions and the 60-second slide presentation for the first condition, subjects were told to await further instructions. Forty-five seconds later instructions for the second condition were presented and following this, subjects were told to await further instructions. The five-minute period preceding these conditions served as the baseline for both fear and anger situations.

The laboratory session lasted approximately 60 minutes. A debriefing followed the end of the session. During this time, subjects' questions were addressed and their subjective evaluations of the emotional stressors were solicited (see Appendix G). Following this, subjects were thanked for their participation and dismissed.

Physiological Data Reduction and Scoring Procedures

The scoring intervals employed in the present study consisted of three 5-minute baseline periods, each of which were followed by a 5-minute relaxation period, an 8-minute mental test, and two 60-second periods designed to provoke fear and anger. Data reduction for these seven scoring intervals was accomplished by dividing each interval into 15-second periods. These intervals were further sub-divided into 5-second segments, one of which was randomly selected and scored to represent the 15-second period. Segments which contained artifacts were deleted from the analysis. Scores for each physiological measure were further reduced such that single values were computed for each of three baselines and four experimental conditions.

In Table 5 is a description of the units of measurement for each of the eight physiological variables employed in this study. The scoring procedures described below were applied to all baseline and stress conditions.

Cephalic muscle tension. Measures of total amplitude muscle activity, to the nearest .5 mm of pen deflection were determined for frontalis and trapezius recordings. Total amplitude for EMG activity levels were obtained by summing the amplitudes of all integrated output ramps occurring during the interval. Mean response measures for

Table 5
Quantification of Physiological Responses

Response System	Channel	Response Mode	Unit of Measurement
Cephalic Musculature	1	Frontalis EMG	mm/sec
	2	Trapezius EMG	mm/sec
Cephalic Vasculature	3	Temporal Artery Blood Pulse Volume	μ v
Respiration	4	Respiration Amplitude	mm
	4	Respiration Frequency	resp/min
Electrodermal	5	Skin Resistance Level (SRL)	k-ohms
	5	Skin Conductance Responses (SCR)	micromhos
Cardiac	6	Heart Rate	beats/min

frontalis and trapezius EMG are expressed in mm/sec.

Cephalic blood pulse volume. Peak-to-trough measures in mm were computed for cephalic vasomotor responses. Response amplitude correlates directly with blood pulse volume such that cephalic vasodilation is indicated by increasing values while decreasing values indicate vasoconstriction. Vasomotor responses are expressed in microvolts (μv) such that 1 mm pen deflection equals .5 μv .

Respiration frequency. The number of respiration cycles per minute was estimated by counting the number of cycles in any given 5-second period and multiplying this value by 12. Mean respiration frequency is expressed in respirations/min.

Respiration amplitude. Peak-to-trough measures in mm were computed for respirations.

Skin resistance level. Levels of skin resistance are expressed in k-ohms. Skin resistance levels were recorded at the beginning of and at 1-minute intervals throughout each condition. Positive differences between the first and last measures (pre minus post) for each condition served as indications of decreased resistance (arousal), while negative values corresponded to increased resistance (relaxation).

Skin conductance responses. Skin resistance changes which were greater than 1% of the immediately preceding skin resistance levels were scored and converted to microhmhos for expression as skin conductance responses (SCR). SCR were normalized by taking the square root of each SCR as the final datum.

Heart rate. Cardiac activity was averaged for each 5-second segment, multiplied by 12, and expressed as beats/min.

RESULTS

Analysis of Physiological Data

Analyses of physiological data were of three types. Raw scores for eight physiological measures were analyzed to determine the extent of initial baseline differences among groups. Two subsequent transformations were made on physiological data. The first involved obtaining scores representing the difference between each condition and its preceding baseline. Change scores were employed in the repeated measures ANOVAs for each physiological variable and in the post-hoc Tukey's Honestly Significant Difference (HSD) tests. A second transformation converting change scores to standard scores was performed to facilitate further post-hoc testing using Kendall's coefficient of concordance W.

Baseline. Separate one-way ANOVAs were performed on all physiological response measures to determine whether initial baseline values significantly differed among the three experimental groups. All three baseline measures for the three headache groups are displayed in Table 6. Caution is necessary in interpreting significance from the temporal artery pulse volume baseline measures because this variable reflected relative, not absolute, plethysmographic readings which are influenced by factors such as placement site and pressure (Gannon et al., 1982).

Table 6
Headache Group Physiological Baselines

Variable		Baseline I		Baseline II		Baseline III	
		\bar{X}	SD	\bar{X}	SD	\bar{X}	SD
Frontalis	MIG	2.30	1.08	1.92	1.09	1.39	1.84
EMG	MCH	3.75	3.20	2.51	2.72	2.20	2.16
(mm/sec)	CON	1.59	1.76	1.46	1.80	1.66	1.98
Trapezius*	MIG	2.76*	1.90	2.95*	1.42	2.81	2.84
EMG	MCH	8.48	8.69	8.50	6.90	6.62	6.76
(mm/sec)	CON	2.80	1.99	2.61	1.35	2.74	1.52
Temporal	MIG	20.15	7.95	17.85	7.19	20.50	6.52
Pulse	MCH	15.30	4.54	16.15	4.55	17.30	5.56
Volume (μ v)	CON	20.15	9.81	21.15	8.08	20.20	7.44
Respiration	MIG	14.90	2.09	15.60	3.27	16.56	2.90
Frequency	MCH	13.50	5.26	12.78	4.42	14.46	4.90
(rpm)	CON	15.24	2.67	15.00	3.58	13.98	2.26
Respiration	MIG	51.00	25.03	41.50	39.14	45.00	16.58
Amplitude	MCH	47.00	15.33	42.00	27.53	40.00	17.39
(mm)	CON	38.25	19.11	39.50	27.43	42.25	18.95
Skin	MIG	126.2	124.9	138.8	144.2	124.1	133.9
Resistance	MCH	78.3	40.5	98.9	106.9	110.3	139.5
Level (k-ohm)	CON	214.7	238.7	209.4	216.1	193.8	214.5
Skin (μ mho)	MIG	.14	.32	.05	.15	.15	.34
Conductance	MCH	.04	.19	.15	.34	.35	.53
Response	CON	.00	.17	.10	.42	.27	.41
Heart	MIG	75.4	9.4	74.8	8.3	75.1	9.4
Rate	MCH	76.2	6.5	74.1	7.5	76.8	5.2
(bpm)	CON	74.3	12.5	75.5	12.5	74.8	13.1

*Group effect, $p < .05$.

Significant baseline differences among groups on trapezius EMG were indicated by the ANOVAs for the first and second baselines, $F(2, 29) = 2.912, p < .05$, and $F(2, 29) = 2.661, p < .05$, respectively. Tukey's HSD tests, however, did not indicate which group means were significantly different from each other. Repeated measures ANOVAs performed on baseline data indicated no effect of trials for any of the physiological variables.

Stress condition order and emotional stress order. Separate univariate analyses of differences between groups of subjects in six treatment order conditions were performed for each physiological variable. Results indicated no significant effects for stress condition order and thus indicate that carry-over effects from one condition to another were inconsequential. Similar analyses performed on differences between two groups receiving initial exposure to either fear or anger yielded no significant order effects.

Emotional stressor (fear and anger) differentiation. Differences between fear and anger responses were tested using paired t -tests on each physiological variable. Results displayed in Table A (see Appendix J) indicate that the fear and anger conditions were not physiologically differentiated for the overall sample for any of the eight physiological measures.

Effects of experimental conditions. Eight repeated measures ANOVAs were performed on physiological data to determine the effects of experimental conditions. Data presented in Table 7 indicate significant effects for stress conditions in six of the eight physiological measures (frontalis and trapezius EMG, temporal artery pulse volume,

Table 7
Summary Tables for Analysis of Headache Group
Physiological Responses Across Experimental Conditions

Variable	Sources of Variation	df	F
Frontalis EMG	Group	2	2.624
	Condition	3	3.857*
	Group x Condition	6	1.818
Trapezius EMG	Group	2	.415
	Condition	3	3.712*
	Group x Condition	6	1.049
Temporal Artery Pulse Volume	Group	2	2.281
	Condition	3	8.786**
	Group x Condition	6	1.490
Respiration Frequency	Group	2	3.533*
	Condition	3	5.686**
	Group x Condition	6	5.387**
Respiration Amplitude	Group	2	1.196
	Condition	3	4.642*
	Group x Condition	6	1.794
Skin Resistance Level	Group	2	.582
	Condition	3	1.260
	Group x Condition	6	.455
Skin Conductance Response	Group	2	.561
	Condition	3	1.211
	Group x Condition	6	.869
Heart Rate	Group	2	.162
	Condition	3	15.170**
	Group x Condition	6	1.310

* $p < .05$.

** $p < .01$.

respiration rate and amplitude, and heart rate). The Group X condition interaction for respiration frequency was also significant ($p < .001$) and is displayed in Figure 4. The main effect of conditions for respiration amplitude was significant ($p < .05$). Details of the relationships among the experimental conditions and physiological responses are described in the following sections, in Tables 7-10, and in Figures 1-8.

Tables 8 and 9 and Figures 1-8 indicate the change scores from baseline group means for each measure during relaxation, mental test, and the fear and anger conditions. Negative values in Tables 8 and 9 and in Figures 1-8 indicate decreases in activity compared to baseline, while positive values corresponded to activity levels which were greater than baseline values. For all variables except skin resistance level and skin conductance response, increases in activity are associated with increased autonomic arousal. For skin resistance levels and skin conductance responses, negative values in Table 9 and Figures 7 and 8 are indicative of increased autonomic arousal.

Post-hoc analyses were performed on the six physiological variables found to significantly differ across experimental conditions. Tukey's HSD tests on frontalis and trapezius EMG data revealed that the relaxation means were significantly lower than means of the fear and anger conditions. Tukey's HSD tests of temporal artery pulse volume data indicated that the relaxation mean was lower than each of the other conditions--mental test, fear, and anger. Tukey's HSD analysis of respiration frequency differences across conditions indicated that the mental test mean was higher than the relaxation mean. For

Table 8
Group Means of Change Scores from Baseline
Across Experimental Conditions for Predicted Variables

Conditions	Physiological Measures					
	Frontalis EMG ^b		Trapezius EMG ^b		Temporal Artery Pulse Volume ^a	
	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD
Relax						
MIG	-.81	1.65	.68	1.20	.18	5.88
MCH	-1.16	2.02	-.96	1.85	.27	1.60
CON	-.62	.96	.28	.53	-1.52	3.17
Mental Test						
MIG	.52	.67	.56	.91	4.02	6.89
MCH	1.70	1.49	.71	1.63	2.62	6.59
CON	.61	.55	.46	.88	4.02	6.15
Stress-Fear						
MIG	.87	.92	1.41	2.08	7.82	7.79
MCH	2.73	4.06	5.61	8.87	1.15	2.61
CON	.37	.58	.83	1.17	5.95	6.67
Stress-Anger						
MIG	.81	.72	1.74	2.16	8.20	5.72
MCH	2.06	3.49	3.07	4.92	2.40	2.91
CON	.32	.50	.60	1.05	5.05	5.88

^aMain effect of experimental condition, $p < .01$.

^bMain effect of experimental condition, $p < .05$.

Table 9

Group Means of Change Scores from Baseline
Across Experimental Conditions for Unpredicted Variables

Conditions	Physiological Measures									
	Respiration ^{a,c,d}		Respiration ^b		Skin		Skin		Heart ^a	
	Frequency		Amplitude		Resistance		Conductance		Rate	
	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD	\bar{X}	SD
Relax										
MIG	1.76 ^c	1.99	-5.33	13.58	-8.29	25.39	-.14	.25	-.97	4.30
MCH	-1.14	3.04	1.33	11.61	17.09	93.13	-.04	.13	-1.75	2.19
CON	-2.20	2.67	5.92	14.12	-14.16	32.02	.00	.04	-1.80	4.43
Mental Test										
MIG	-.16	3.77	13.67	10.94	47.87	104.77	-.06	.22	5.57	5.12
MCH	3.18	3.26	-.33	16.39	-20.83	92.90	-.10	.21	9.58	9.48
CON	2.92	2.83	5.00	9.87	-45.77	75.19	-.04	.14	8.82	6.59
Stress-Fear										
MIG	-2.97 ^c	3.93	33.38	39.71	-39.96	84.86	-.04	.13	6.15	7.06
MCH	-1.38	4.07	22.38	37.30	-22.84	79.96	.00	.23	4.20	7.23
CON	2.04	2.34	18.75	36.68	-14.28	49.92	-.04	.19	4.41	9.16
Stress-Anger										
MIG	-3.78 ^c	5.06	30.00	41.98	46.10	107.82	-.03	.14	7.32	7.51
MCH	.24	3.27	17.00	34.74	-28.59	104.13	-.01	.03	2.88	7.12
CON	.99	1.99	15.25	30.75	-17.18	59.52	-.05	.14	2.43	4.93

^aMain effect of experimental condition, $p < .01$.

^bMain effect of experimental condition, $p < .05$.

^cMain effect of group, $p < .05$.

^dInteraction of group by experimental condition, $p < .05$.

Table 10

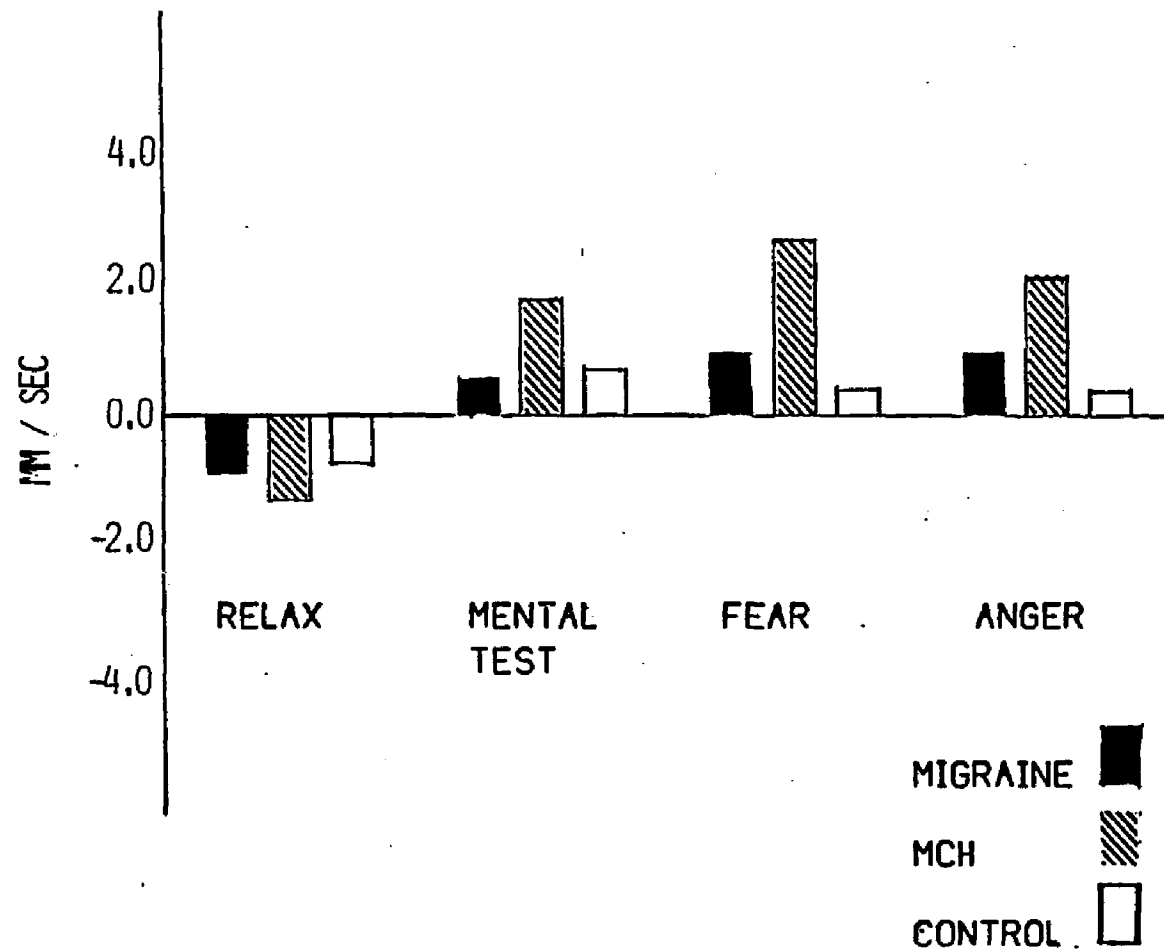
A Priori Contrasts for Headache Group Physiological Differences
Across Three Stress Conditions

Variables	Predictions	Contrasts	Stress Conditions					
			Mental Test		Fear		Anger	
			t ⁺	p	t ⁺	p	t ⁺	p
Frontalis EMG	MCH > MIG > CON	MIG - MCH	-.453	.658	-1.332	.216	-1.737	.113
		MIG - CON	.581	.568	-1.210	.216	1.283	.220
		MCH - CON	.803	.438	-1.489	.171	1.156	.064
Trapezius EMG	MCH > MIG > CON	MIG - MCH	1.476	.166	-.412	.687	.163	.873
		MIG - CON	-.056	.956	-.385	.719	.340	.738
		MCH - CON	-1.510	.157	.214	.834	.199	.845
Temporal Artery Pulse Volume	MIG > CON > MCH	MIG - MCH	.465	.648	2.568	.026*	2.857	.013*
		MIG - CON	.00	1.00	.578	.570	1.214	.240
		MCH - CON	-.491	.629	-2.119	.056	-1.277	.224

*p < .05.

⁺df = 1,19.

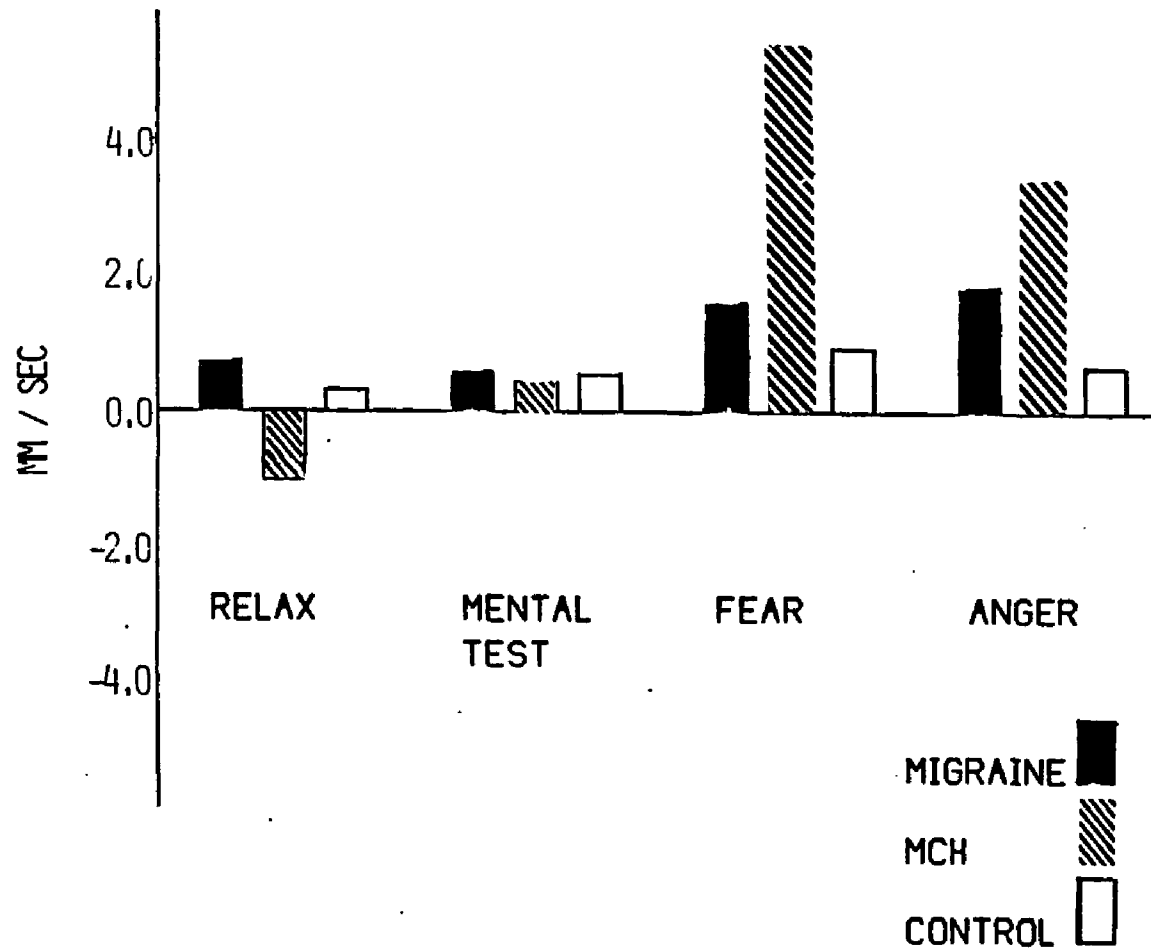
FIGURE 1
FRONTALIS EMG GROUP MEANS^b



^bMain effect of experimental condition, $p < .05$.

FIGURE 2

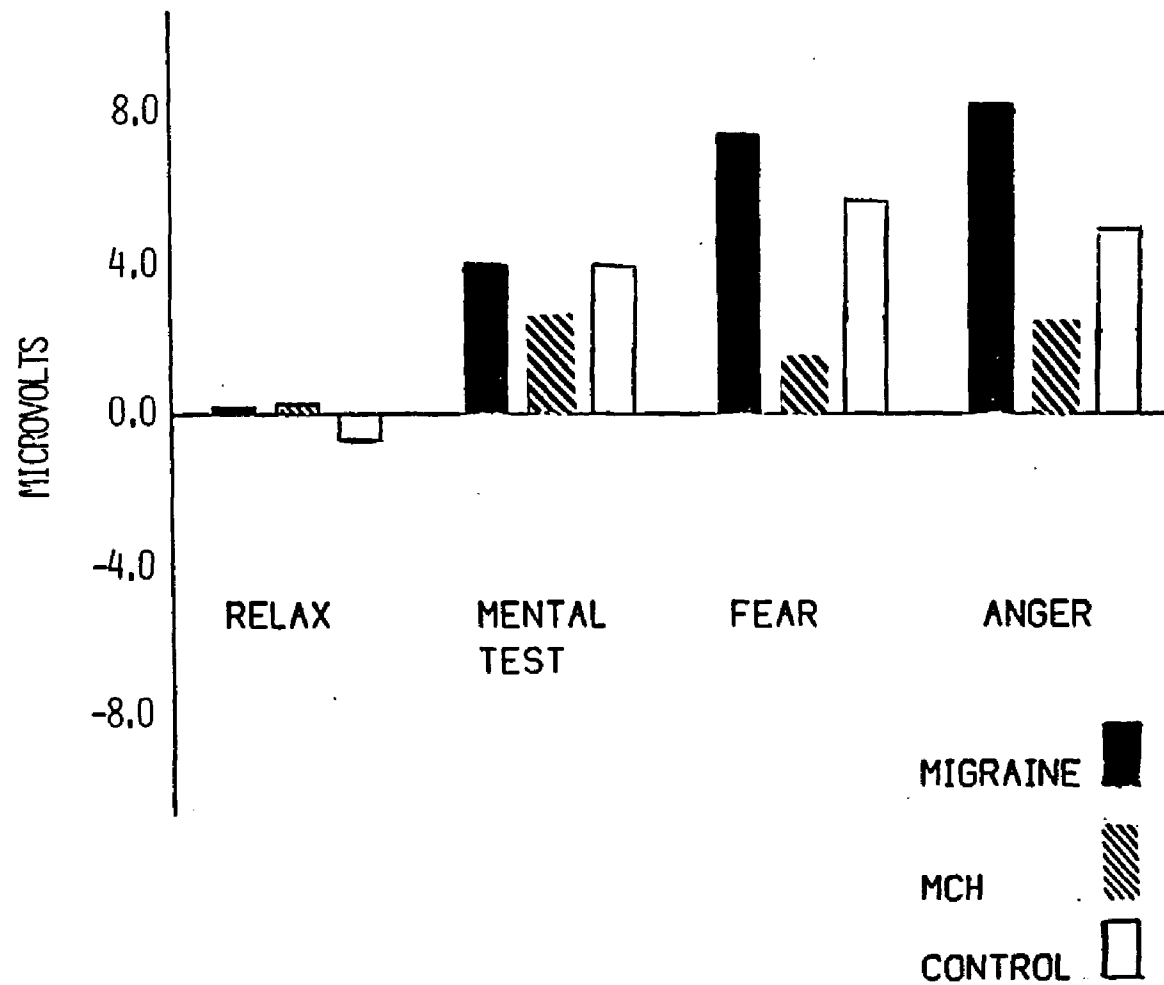
TRAPEZIUS EMG GROUP MEANS^b



^bMain effect of experimental condition, $p < .05$.

FIGURE 3

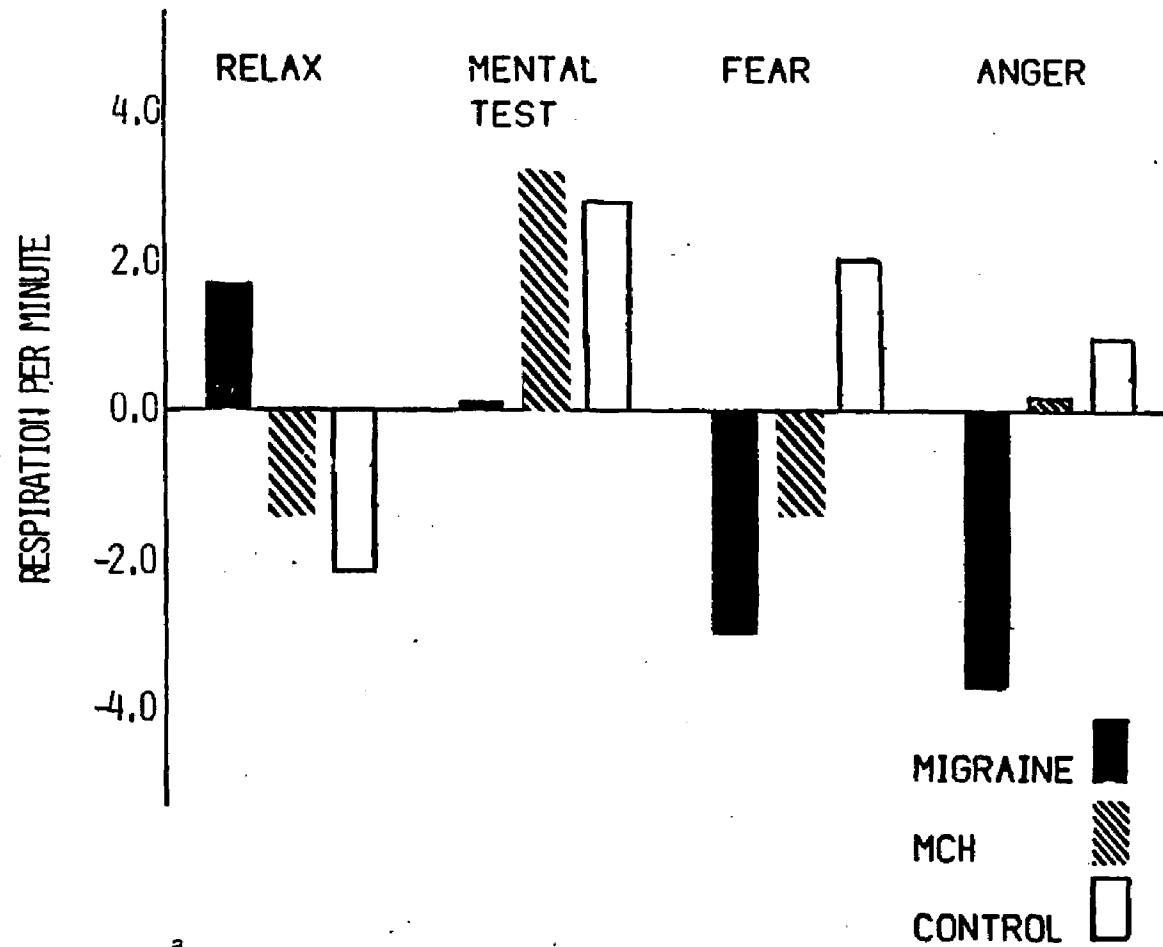
TEMPORAL PULSE VOLUME GROUP MEANS^a



^aMain effect of experimental condition, $p < .01$.

FIGURE 4

RESPIRATION FREQUENCY GROUP MEANS^{a,c,d}



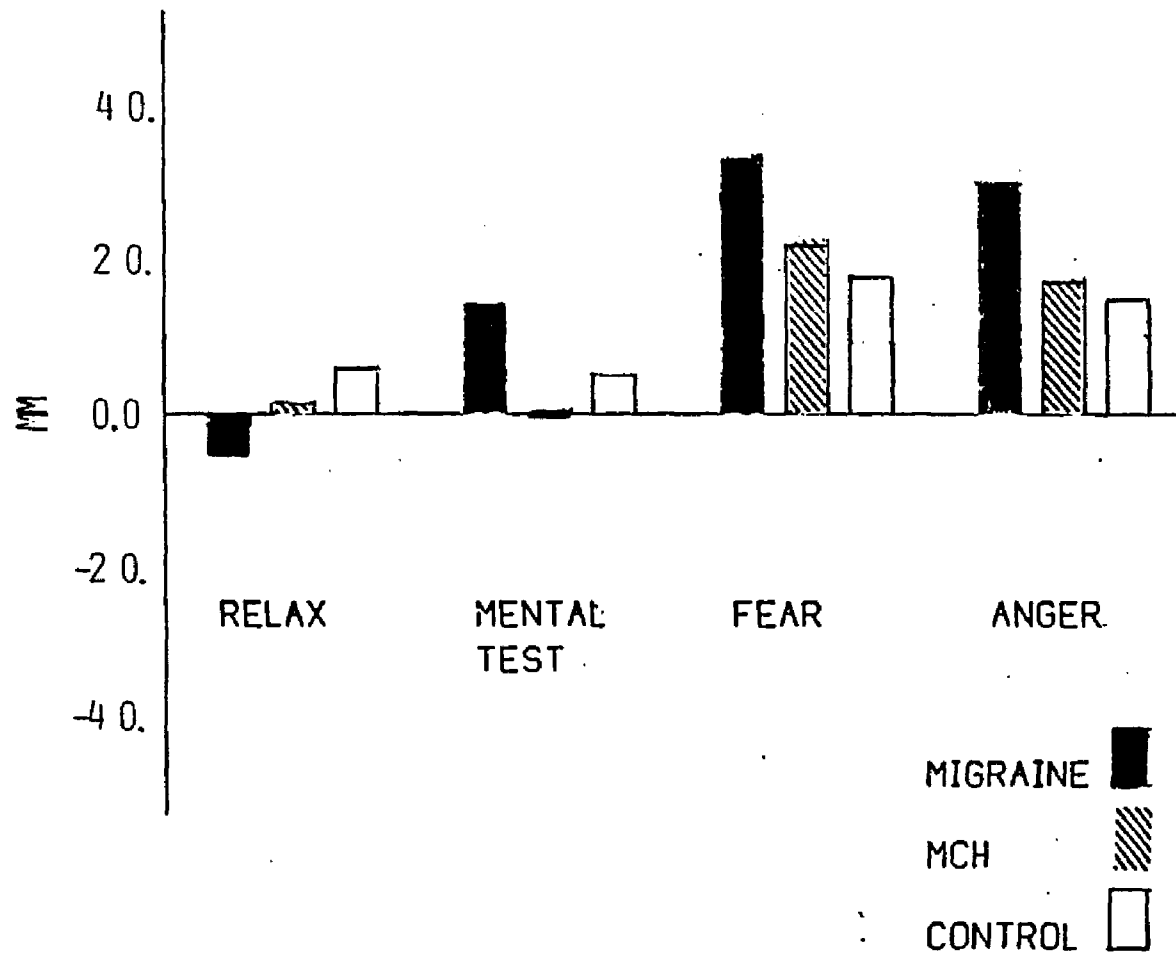
^aMain effect of experimental condition, $p < .01$.

^cMain effect of group, $p < .05$.

^dInteraction of group by experimental condition, $p < .05$.

FIGURE 5

RESPIRATION AMPLITUDE GROUP MEANS^b



^bMain effect of experimental condition, $p < .05$.

FIGURE 6

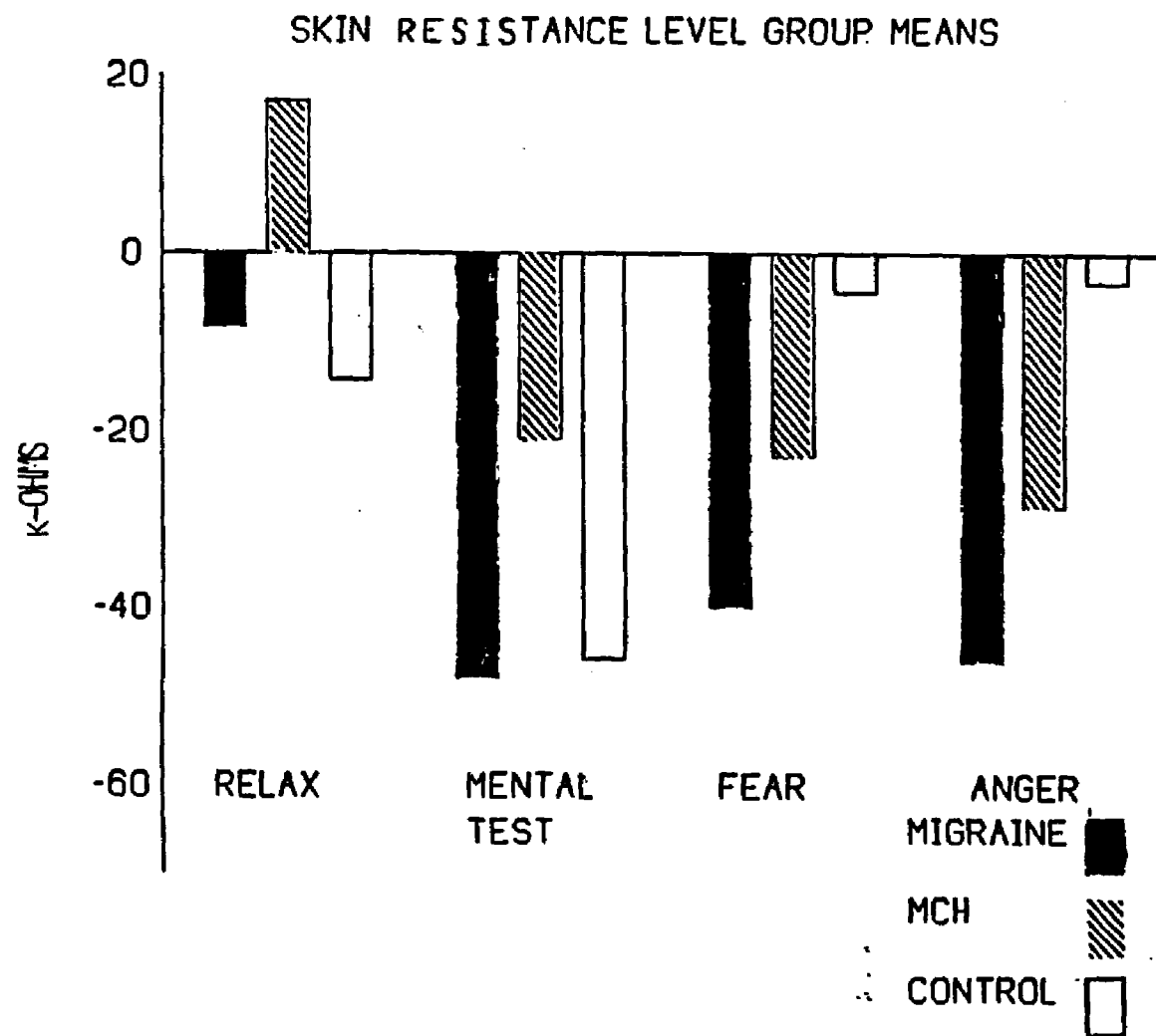


FIGURE 7

SKIN CONDUCTANCE RESPONSE GROUP MEANS

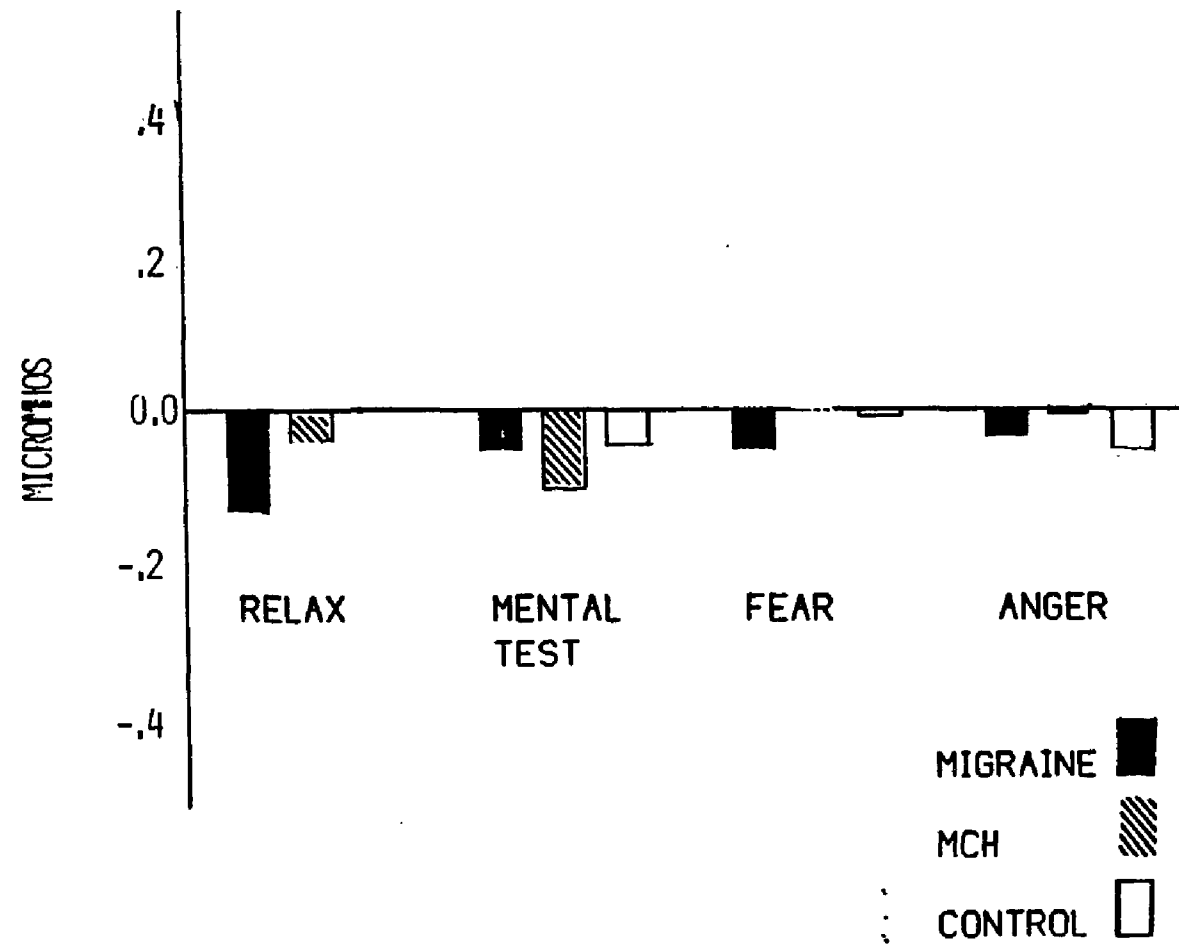
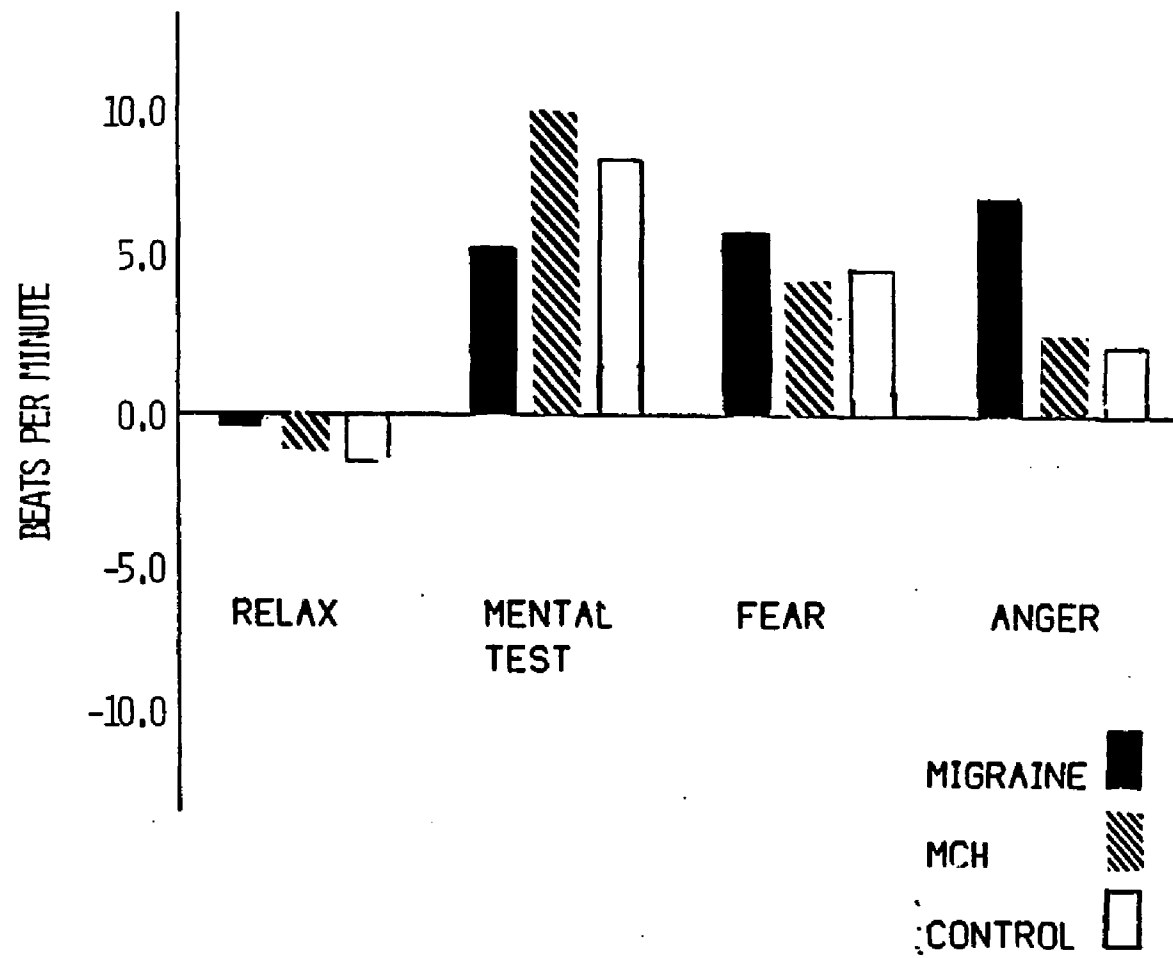


FIGURE 8

HEART RATE GROUP MEANS^a



^aMain effect of experimental condition, $p < .01$.

respiration amplitude, Tukey's HSD tests indicated that the relaxation mean was lower than both the fear and anger means. Finally, a similar post-hoc analysis of heart rate data revealed that the relaxation mean was lower than all other conditions.

Individual experimental conditions. Group means for change scores from baseline for each physiological variable across experimental conditions are presented in Tables 8 and 9. Table 8 presents data for three physiological variables for which experimental predictions were made--frontalis and trapezius EMG and temporal artery pulse volume. Data for the remaining five variables are depicted in Table 9. The effect of groups across conditions of relaxation, mental test and emotional stressor (fear and anger) for each physiological variable is also depicted in Tables 8 and 9. These comparisons are also shown in Figures 1-8. Respiration rate emerged as the only variable associated with group differences, $F(2, 29) = 3.532, p < .05$. The Group X experimental condition interaction was also significant, $F(6, 29) = 5.380, p < .01$.

During relaxation, differences among groups were observed only for respiration frequency, $F(2, 29) = 5.882, p < .05$. Migraineurs demonstrated slight increases in respiration rate from baseline, while MCH subjects evidenced negligible decreases, and controls showed greater decreases. Tukey's HSD tests indicated that significant differences existed only between the migraine and control groups.

For the mental test condition, no significant group differences were observed for any of the eight physiological measures.

Although fear and anger were not found to be significantly differentiated from each other on the physiological measures, they were examined individually with respect to their effects on physiological functioning across the experimental and control groups. For the fear condition, respiration rate was the only variable found to differ significantly among groups, $F(2, 29) = 5.117$, $p < .05$. Compared to the baseline immediately preceding the fear condition, migraineurs reduced their respiration rates, MCH subjects demonstrated negligible reductions, and controls showed increased rates of respiration. Tukey's HSD tests revealed that significant differences existed only between migraine and control means.

Very similar patterns to those described for fear occurred in the anger condition (see Figure 5). Respiration rate was again the only variable significantly distinguishing among groups, $F(2, 29) = 4.887$, $p < .05$. Tukey's HSD tests identified significant differences between the migraine group's reductions in respiration rate and the control group's increased breathing rate.

Headache symptom-specificity. Experimental predictions related to migraine and MCH symptom-specificity were tested using planned contrast procedures. Table 10 describes the a priori relationships hypothesized between headache groups under stress and three cephalic measures including frontalis EMG, trapezius EMG, and temporal artery pulse volume. Results of contrast analyses are also listed in Table 10.

Migraine symptom-specificity was partially supported by the planned contrast analyses which were significant for only temporal artery pulse volume data. Migraineurs were significantly more dilated

than MCH subjects during the fear and anger conditions, $t(1, 19) = 2.568$, $p < .05$, $t(1, 19) = 2.857$, $p < .05$, respectively. Similar comparison between migraine and control groups and MCH and controls did not yield significant differences.

Hypotheses related to MCH symptom-specificity were not supported. Levels of frontalis and trapezius EMG activity were not significantly greater for MCH subjects than for either the migraine or the control group.

Analyses of individual-response stereotypy. The hypotheses related to migraine and MCH stereotypy were tested using additional post-hoc analyses. Quantification of physiological response patterning across stress conditions for headache groups was accomplished with Kendall's coefficient of concordance W . The extent to which variables demonstrate stable rank orderings across conditions is represented by this value. Totally random patterns of physiological functioning across various tasks would yield a concordance W value of 0; fixed patterns represent the other end of the continuum and would yield a value of 1.

For the purpose of this analysis subjects' change scores were converted to standard scores for each variable and analyzed to obtain individual concordance W values. Headache group scores were analyzed in the same fashion to yield group concordance W values. Figures 9-11 illustrate the physiological patterns associated with the four stress conditions for groups of migraineurs and MCH and control subjects respectively. None of the headache groups yielded significant concordance W values. The coefficient of concordance W was highest for

FIGURE 9

MIGRAINE RESPONSE PATTERNING

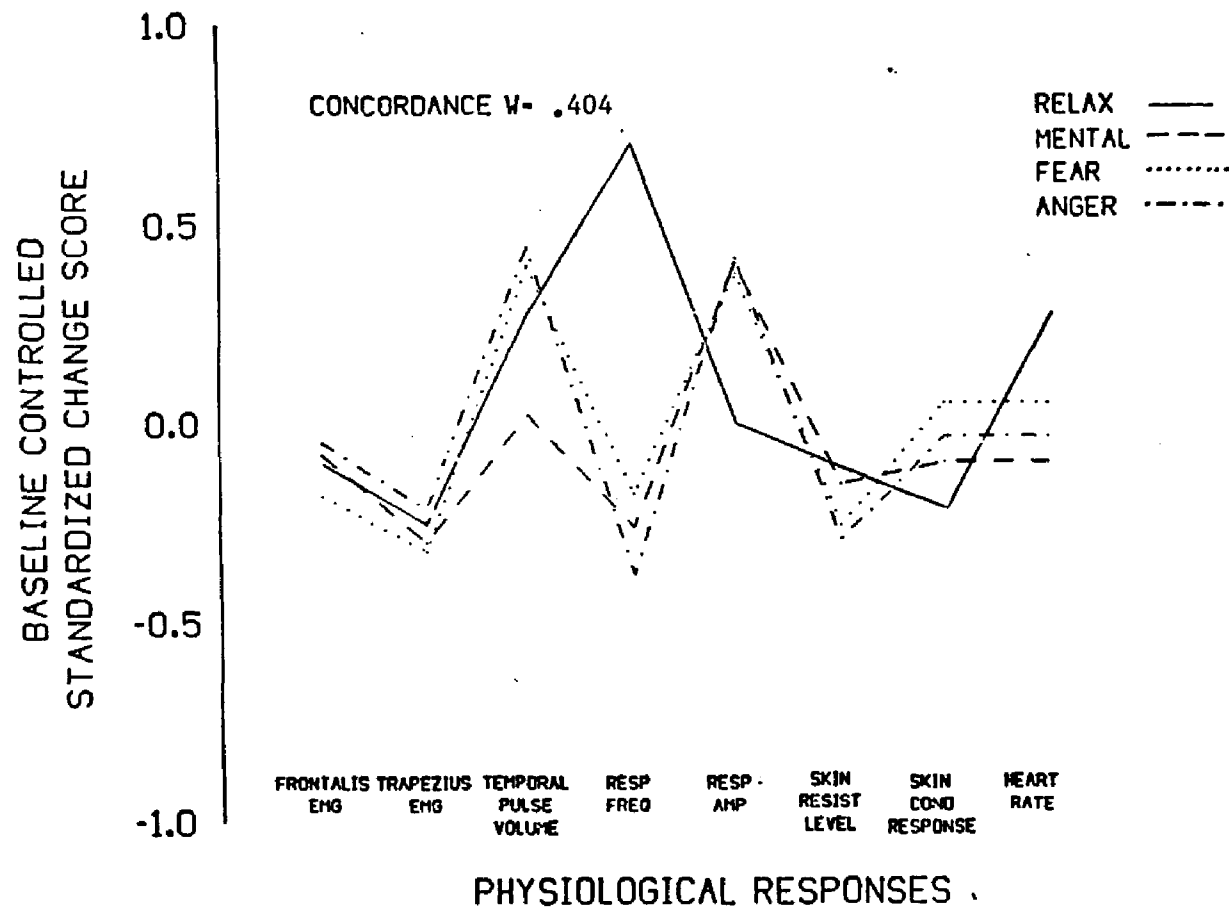


FIGURE 10

MCH RESPONSE PATTERNING

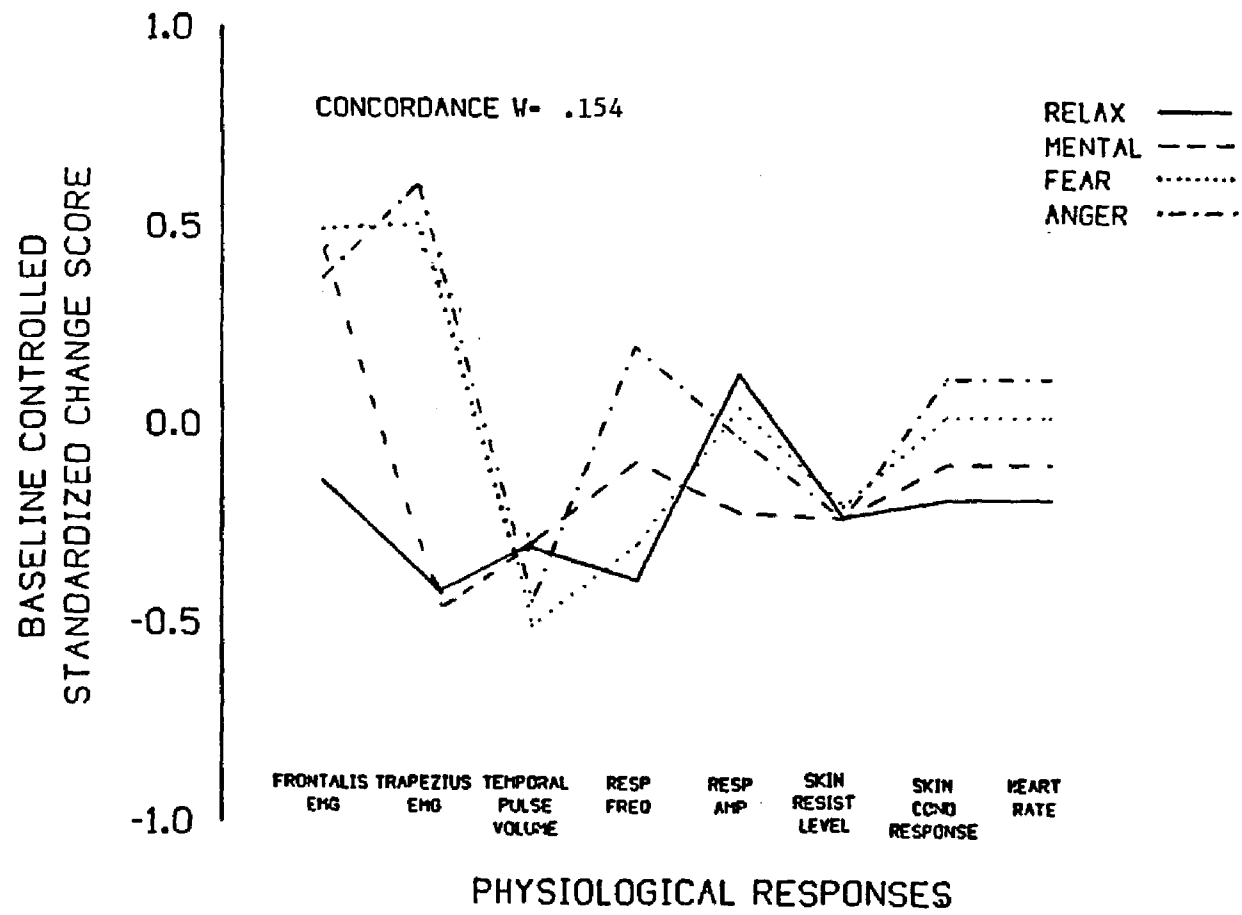
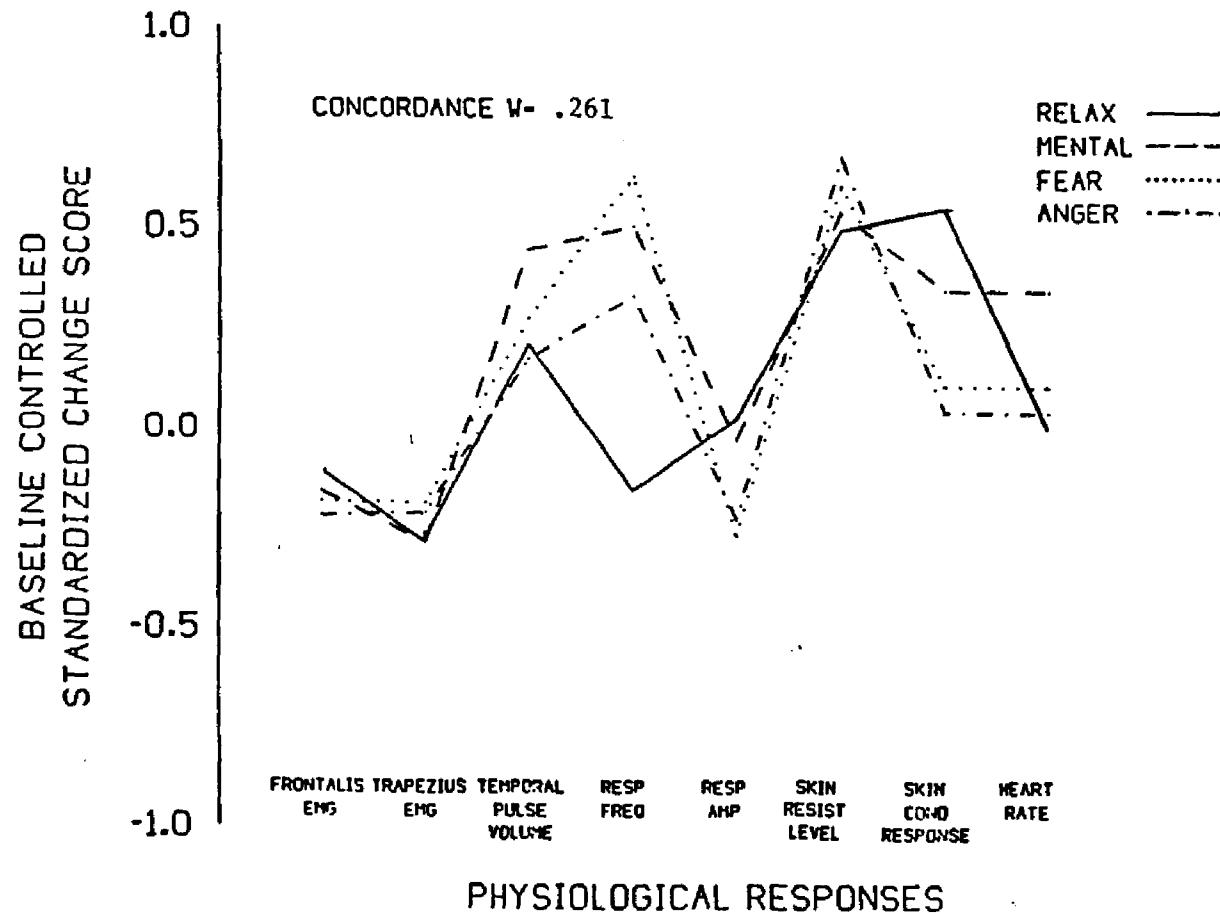


FIGURE 11

CONTROL RESPONSE PATTERNING



the migraine group ($W = .405$) but not statistically significant, Friedman's X^2 statistic (7) = 11.333, $p > .05$. The control group concordance W value ($W = .261$) was also not significant, Friedman's X^2 statistic (7) = 7.333, $p > .05$. Finally, the MCH group yielded the lowest concordance W value ($W = .155$), Friedman's X^2 statistic (7) = 4.333, $p > .05$. These findings indicate no support for response stereotypy among headache groups or controls.

Analysis of Psychological Data

Means for self-report scales are displayed in Table 11. One-way ANOVAs were performed on 12 subscales of five self-report measures. Results indicated that group differences were significant for six of the self-report measures--MAACL Hostility ($p < .05$); Life Change Scale, quantitative and qualitative stress measure ($p < .05$ and $p < .01$, respectively); Levenson's Scale related to internality ($p < .01$); and SUDS Scales associated with distress ratings of fear and anger conditions ($p < .05$, $p < .05$, respectively). Subsequent post-hoc analyses using Tukey's HSD tests were performed on measures yielding significant simple main effects. Turkey's HSD tests indicated that MCH subjects reported more MAACL Hostility and higher SUDS ratings during fear and anger than the controls. Controls reported significantly more internality than MCH subjects. In regard to the Life Change Scale, Tukey's tests revealed that both the migraine and MCH groups reported experiencing stresses of greater quantity and quality compared to controls.

Table 11
Headache Group Means and Probabilities for Self-Report Scores

Self-report Measure	Group			p
	Migraine	MCH	Control	
Beck Depression Inventory	29.1	30.0	27.5	.544
Multiple Affective Adjective Checklist (MAACL)				
Anxiety	8.1	10.8	7.9	.319
Depression	11.8	17.3	13.1	.179
Hostility	9.1	14.9	8.3	.022*
Life Change Scale				
Quantity	250.3	251.9	138.5	.012*
Recency	2.3	2.3	1.4	.097
Quality	3.2	3.3	2.2	.005**
Levenson's Scale				
Internal	36.7	31.5	39.2	.005**
Chance	20.5	27.5	20.8	.056
Powerful others	22.2	24.9	20.6	.193
SUDS Rating				
Fear condition	7.4	8.6	5.4	.020*
Anger condition	7.9	8.7	5.5	.016*

* $p < .05$.

** $p < .01$.

The results from the Emotion Survey Form (Appendix G) revealed that the experimental induction of fear and anger were not successful. Table B (See Appendix K) depicts the frequencies with which headache groups endorsed labels of their emotional experiences during the fear and anger conditions. Sorrow emerged as the most frequently endorsed emotion experienced by the sample as a whole in response to both conditions.

Psychophysiological relationships. A final consideration of the present study involved the investigation of relationships among certain physiological variables (responsivity of cephalic vascular and muscular measures) and psychological variables. A matrix generated by correlating selected physiological and all psychological measures is presented in Table C (see Appendix L). The results of this analysis do not offer support for significant relationships among psychological and physiological measures.

DISCUSSION

The present study examined numerous psychological and physiological aspects of headache. For the purpose of clarity, these findings will be discussed as they relate to the specific topics presented in the Introduction.

Methodological Considerations

Several methodological issues were addressed by the present study. The effects of different experimental stressors on physiological functioning of headache groups were compared. The mental test was differentiated from relaxation for temporal artery pulse volume, respiration frequency and heart rate. Although commonly used as a stressful exercise in physiological studies, the mental test did not appear as potent as the emotional stressors in producing autonomic arousal and demonstrating physiological differentiation among headache groups. The fear and anger conditions were differentiated from relaxation for both frontalis and trapezius EMG, respiration amplitude, heart rate, and temporal pulse volume. Additionally, the emotional stressors were instrumental in eliciting the higher degree of cephalic dilation that was observed for the migraine group compared to the MCH group. The results of the present study confirm the hypothesis that emotional stressors are more effective than mental tests in eliciting levels of physiological activation necessary to differentiate physiological functioning among headache groups. The recommendations

made by numerous headache researchers (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Cohen et al., 1982; Gannon et al., 1982; Sturgis, Note 2) to employ laboratory stressors which are adequately stressful appear to be well founded.

Although the fear and anger manipulations employed were not associated with subjects' subjective experience of these specific emotions, most subjects acknowledged that they found the exposure to the slides of human traffic fatalities very stressful. The experimental hypothesis predicting differential physiological responses to fear and anger was neither confirmed nor, perhaps, adequately tested. The failure of the attempts to induce in headache groups the specific emotions of fear and anger may have been due to individuals' powerful reactions to the stimuli which served to override the experimental instructions. Additionally, the experimental instructions employed in the present study may not have been explicit enough to facilitate the experiences of fear and anger. Further attempts to examine the effects of specific emotion on the physiology of headache groups might benefit from employing more carefully controlled stimulus presentations (e.g., films with content specifically related to the emotional experiences of fear, sorrow, anger, mirth, etc.). The manipulation of fear and anger by Ax (1953) and the correlation of physiological responses and emotional reactions in children viewing the film Bambi (Sternbach, 1962) represent creative uses of emotion-specific stimuli. Although these emotion-specific manipulations have yet to be applied in headache psychophysiological research, results of these approaches may prove interesting.

The present study also controlled for carry-over effects by randomly presenting the order of experimental conditions. The results indicated that the effects of stress condition order were inconsequential and confirm a previous report by Gannon et al., 1982.

Migraine and MCH-Symptom Specificity

Hypotheses related to migraine and MCH symptom-specificity received limited support. Differences in initial baselines among experimental groups and controls were significantly different for only trapezius EMG responses, but post-hoc testing could not identify differences between group means. Higher resting levels of EMG activity among headache groups compared to controls have frequently been reported (Acosta et al., 1978; Anderson & Franks, 1981; Bakal & Kaganov, 1977a; and others).

Under conditions of stress, no evidence for MCH symptom-specificity was noted. A priori predictions of increased frontalis and trapezius EMG activity for MCH subjects compared to migraine and control groups were not supported. Predictions of migraine-specificity were partially confirmed. Migraine subjects were found to be significantly more dilated than the MCH group during the emotional stress condition. However, temporal artery blood flow responses during emotional stress conditions revealed significant differentiation only between the migraine and MCH groups but not between migraine and controls. The original reports of migraine-specificity between migraineurs and controls (Tunis & Wolff, 1953) have not been supported by the few researchers who have investigated cephalic blood flow in headache groups (Cohen et al., 1982; Feuerstein et al., 1982; Sturgis,

Note 2) or by the findings of the present study. Possibly, the procedures employed by studies to date to induce emotional arousal were not strong enough to elicit evidence for migraine symptom-specificity. Other explanations for these inconclusive results include the small sample size employed in the present study, as well as the possibility that migraine symptom-specificity may not exist. Future investigations of migraine symptom-specificity which measure cephalic blood flow and expose larger samples of migraineurs to intense stressors may clarify the specificity question in migraine.

Physiological Differentiation between Migraine and MCH

The present study offers considerable evidence suggesting that migraine and MCH groups can be differentiated on the basis of their physiological responses. The data confirmed the experimental prediction related to migraine-MCH differentiation. During fear and anger conditions, migraineurs demonstrated significantly more cephalic dilation than did MCH individuals. These differential patterns of cephalic vascular activity between migraine, control, and MCH groups have also been reported by Tunis and Wolff (1953, 1954). Respiration rates also differed between headache groups. Compared to controls, relaxation and emotional stressors produced markedly greater increases and decreases, respectively, in respiration frequency in migraineurs but not MCH subjects. The implications of respiration rate differences between headache groups are not clear. Replications of these findings are necessary before they can be adequately interpreted. Generally, these findings do not offer support for the position offered by Bakal and Kaganov (1977b) and Cohen (1978) proposing that migraine and MCH

are different quantitative manifestations of the same disorder.

Response Stereotypy

The determination of concordance W values for the three groups provided no support for significant response patterning. Although migraine stereotypy has been previously demonstrated (Cohen et al., 1978; Cohen et al., 1982; Gannon et al., 1982), results from the present study failed to confirm this finding. The limited number of subjects in the sample coupled with the relatively few significant differences between groups on various response measures may explain the inability of the present study to demonstrate migraine stereotypy. Previous researchers (Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Fuerstein et al., 1982; Sturgis, Note 2) have also failed to document evidence supporting migraine stereotypy.

It was suggested by Sternbach (1972) that response stereotypy may contribute to the development of psychosomatic disorders. Manifestations of migraine stereotypy have included rigid responses patterns (Cohen et al., 1978; Cohen et al., 1982) and heightened cephalic vascular reactivity to stress and rigid patterns in migraineurs during recovering periods following stress (Gannon et al., 1982). Future investigations of migraine stereotypy may serve to clarify the treatment rationale for migraine interventions which are aimed at reducing autonomic reactivity via relaxation training (Blanchard et al., 1978; Warner & Lance, 1975; Williamson, 1981) or addressed at specific attempts to modify the reactivity of the cephalic vasculature with cephalic blood flow biofeedback procedures (Allen &

Mills, 1982; Feurerstein & Adams, 1977; Friar & Beatty, 1976). Further empirical testing of response stereotypy using large samples of migraine sufferers is clearly warranted.

Psychological Differences Among Headache Groups

The present study partially confirmed previous reports indicating that headache groups can be distinguished from controls by their patterns of self-report on physiological measures. The finding that a continuum of self-reported distress exists along which controls reported lowest levels of distress and that MCH subjects typically reported the most distress (Andrasik, Blanchard, Arena, Teders, Teven, & Rodichok, 1982; Pratt et al., Note 1) was partially supported by the present study. MCH subjects in the present study were found to yield higher scores than controls on a measure related to hostility and lower scores than controls on a measure of internality. Both migraine and MCH groups' perceptions of stressful life events were greater than those of the non-headache group. Finally, laboratory stressors also were rated as more distressing by the MCH group than by the controls. This last finding is partially at odds with Price and Blackwell's (1980) report that found migraineurs to label an unpleasant film as less distressing than did controls. If headache groups generally perceive events as more stressful than do non-headache groups, a case might be made for incorporating stress management and cognitive-behavioral coping skills (Holroyd & Andrasik, 1980; Mitchel & White, 1977) in headache treatment programs.

Relationships between Psychological and Physiological Measures

The findings from the present study do not suggest that

consistent relationships exist between the self-report measures employed and selected physiological variables. It may be that such relationships do not exist or, alternatively, that the measures employed in the present study are not the most suitable choices for examining such interactions. Future investigations employing larger samples of headache sufferers may be able to address this question more thoroughly.

Summary

The present study indicates that investigations into the multi-determined phenomenon of headache are appropriately addressed by approaches combining psychological and physiological measurements. Based on the results of the present investigation, the following conclusions can be drawn:

1. Migraine and MCH groups can be differentiated from each other based on patterns of cephalic vascular responses. Migraine and control groups were also found to differ with respect to respiration frequency during relaxation and a mental test.
2. MCH symptom-specificity formulations remain generally unsupported, while migraine symptom-specificity received limited support. Response stereotypy was not demonstrated for any of the three groups tested.
3. Laboratory stressors involving emotional distress were more effective in differentiating between headache groups and between headache groups and controls than traditionally employed mental tests. However, attempts to elicit the specific emotions of fear and anger were not successful using the methods described in the present study.

4. Psychological differences between migraine and MCH groups were not found. However, MCH and controls were differentiated on measures related to hostility, internality, and distress ratings associated with experimentally-induced stressors. Both headache groups reported more distress related to life stress than did controls.
5. No support was found for consistent interrelationships between selected psychological and physiological measures.

Future research efforts into the psychophysiology of headache may improve on the present study by including larger samples of headache subjects and by employing stressors which are more specifically linked to the emotions of fear and anger. Ultimately, future investigations of this type will clarify the psychological and physiological contributions to headache and may serve to facilitate more effective treatments of these disorders.

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Appendix A

HEADACHE TELEPHONE QUESTIONNAIRE

Name _____ Phone # _____
Address _____ Time available _____
Interviewer _____ Date _____

1. Describe the pain: pulsing or constant
 mild to moderate or severe and incapacitating
 one-sided or bilateral
 where
2. Do headaches occur with any warning? If so, describe
3. How long does an average headache last?
4. Do any side effects accompany headaches? If so, describe
5. How many headaches do you have a month? Over the last year?
6. What medications are you taking for your headaches?
7. Have you been to a physician for your headaches?
 What was the diagnosis?
 When did you last get medical treatment?
8. What are some events that may be related to your headaches?

Comments:

Headache Classification _____

APPENDIX B
HEADACHE QUESTIONNAIRE

NAME _____ DATE _____

DIRECTIONS: Read each question carefully and then circle the answer which is most correct for you. The 5 possible answers are defined as follows: Always; occurs without exception. Usually; occurs on most occasions with infrequent exceptions. Sometimes; occurs approximately half the time. Infrequently; occurs only once in a great while. Never; absolutely does not occur and has not ever occurred.

	NEVER	INFREQUENTLY	SOMETIMES	USUALLY	ALWAYS
1. I awaken with a headache.	1	2	3	4	5
2. My headache ends within 4 hours.	1	2	3	4	5
3. My headache ends within 6 hours.	1	2	3	4	5
4. My headache ends within 12 hours.	1	2	3	4	5
5. My headache ends within 24 hours.	1	2	3	4	5
6. I have sudden attacks of headache.	1	2	3	4	5
7. My headache is worst at the end of the working day.	1	2	3	4	5
8. My headache is throbbing or pulsating.	1	2	3	4	5
9. My headache feels like a tightness or an external pressures (band-like or cap-like).	1	2	3	4	5
10. My headache begins on the left-hand side of my head.	1	2	3	4	5
11. My headache begins on the right-hand side of my head.	1	2	3	4	5
12. When I get a headache I have visual changes like seeing stars, blind spots, double vision and/or become sensitive to light.	1	2	3	4	5
13. My headache begins in my neck, shoulders or the back of my head.	1	2	3	4	5
14. I have nausea and vomiting with my headaches.	1	2	3	4	5
15. My headache gets worse if I cough, strain, or lift objects.	1	2	3	4	5
16. My headache is better if I can loosen up my neck muscles.	1	2	3	4	5
17. Aspirin, Anacin, Bufferin, Excedrin, BC, Alka Seltzer, or other non-prescription pain medications relieve my headache.	1	2	3	4	5
18. I take a prescribed medication to prevent a full blown attack of a headache.	1	2	3	4	5
19. My headache starts during periods of relaxation.	1	2	3	4	5

ALWAYS
USUALLY
SOMETIMES
RARELY
NEVER

20. My headache begins early in the morning and increases in severity as the day continues.

1 2 3 4 5

21. My headache starts after smoking or drinking coffee or liquor.

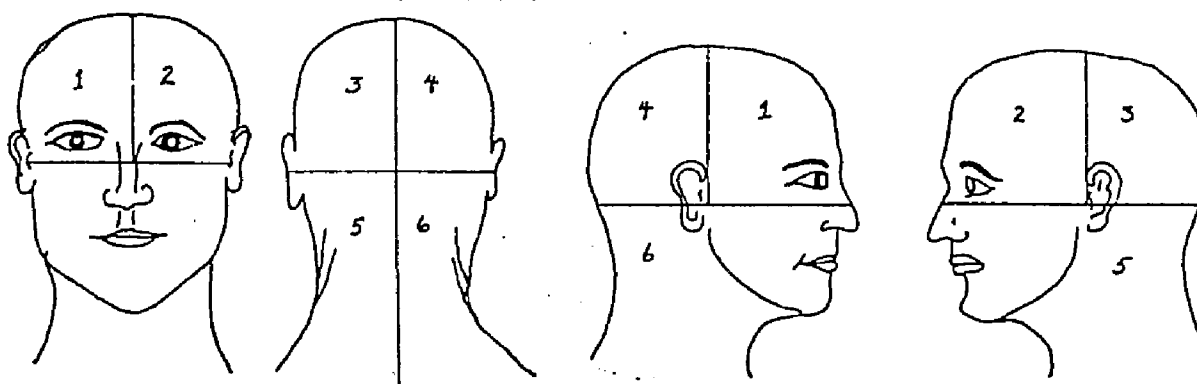
1 2 3 4 5

For the next two questions please refer to the figures below.

22. When I get a headache the most severe pain occurs in area(s) _____.

23. When I get a headache I experience pain in area(s):

only 1	1 2 3 4 5
only 2	1 2 3 4 5
only 3	1 2 3 4 5
only 4	1 2 3 4 5
only 5	1 2 3 4 5
only 6	1 2 3 4 5
only 1 & 2	1 2 3 4 5
only 1 & 4	1 2 3 4 5
only 1 & 5	1 2 3 4 5
only 1, 4 & 6	1 2 3 4 5
only 2 & 3	1 2 3 4 5
only 2 & 5	1 2 3 4 5
only 2, 3, & 5	1 2 3 4 5
only 3 & 4	1 2 3 4 5
only 1, 2, 3 & 4	1 2 3 4 5
only 1, 2, 3, 4, 5 & 6	1 2 3 4 5



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APPENDIX C

L.C. SCALE

Directions:

For each event you have experienced within the last two years place a check in the appropriate time column.

For each event checked rate the degree of necessary adjustment to this event on a scale of 1 to 5 where 1 represents relatively easy adjustment and 5 represents maximally difficult adjustment.

EVENTS	TIME				RATING
	0-6 mos	7-12 mos	12-18 mos	19-24 mos	
Family:					
Death of spouse	_____	_____	_____	_____	_____
Divorce	_____	_____	_____	_____	_____
Marital separation	_____	_____	_____	_____	_____
Death of close family member	_____	_____	_____	_____	_____
Marriage	_____	_____	_____	_____	_____
Marital reconciliation	_____	_____	_____	_____	_____
Major change in health of family	_____	_____	_____	_____	_____
Pregnancy	_____	_____	_____	_____	_____
Addition of new family member	_____	_____	_____	_____	_____
Major change in arguments with spouse	_____	_____	_____	_____	_____
Son or daughter leaving home	_____	_____	_____	_____	_____
In-law troubles	_____	_____	_____	_____	_____
Spouse starting or ending work	_____	_____	_____	_____	_____
Major change in family get togethers	_____	_____	_____	_____	_____
Personal:					
Detention in jail	_____	_____	_____	_____	_____
Major personal injury or illness	_____	_____	_____	_____	_____
Sexual difficulties	_____	_____	_____	_____	_____
Death of a close friend	_____	_____	_____	_____	_____
Outstanding personal achievement	_____	_____	_____	_____	_____
Start or end of formal schooling	_____	_____	_____	_____	_____
Major change in living conditions	_____	_____	_____	_____	_____
Major revision of personal habits	_____	_____	_____	_____	_____
Changing to a new school	_____	_____	_____	_____	_____
Change in residence	_____	_____	_____	_____	_____
Major change in recreation	_____	_____	_____	_____	_____
Major change in church activities	_____	_____	_____	_____	_____
Major change in sleeping habits	_____	_____	_____	_____	_____
Major change in eating habits	_____	_____	_____	_____	_____
Vacation	_____	_____	_____	_____	_____
Christmas	_____	_____	_____	_____	_____
Minor violations of the law	_____	_____	_____	_____	_____
Work:					
Being fired from work	_____	_____	_____	_____	_____
Retirement from work	_____	_____	_____	_____	_____
Major business adjustment	_____	_____	_____	_____	_____
Changing to different line of work	_____	_____	_____	_____	_____
Major change in work responsibilities	_____	_____	_____	_____	_____
Trouble with boss	_____	_____	_____	_____	_____
Major change in working conditions	_____	_____	_____	_____	_____
Financial:					
Major change in financial state	_____	_____	_____	_____	_____
Mortgage or loan over \$10,000	_____	_____	_____	_____	_____
Mortgage foreclosure	_____	_____	_____	_____	_____
Mortgage or loan less than \$10,000	_____	_____	_____	_____	_____

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APPENDIX D

BECK INVENTORY

Choose one statement for each item which best describes how you feel most of the time and circle the number beside it.

- A. 1. I do not feel sad.
2. I feel blue or sad.
3. I am blue or sad all the time and I can't snap out of it.
4. I am so sad or unhappy that it is very painful.
5. I am so sad or unhappy that I can't stand it.
- B. 1. I am not particularly pessimistic or discouraged about the future.
2. I feel discouraged about the future.
3. I feel I have nothing to look forward to.
4. I feel that I won't ever get over my troubles.
5. I feel that the future is hopeless and that things cannot improve.
- C. 1. I do not feel like a failure.
2. I feel I have failed more than the average person.
3. I feel I have accomplished very little that is worthwhile or that means anything.
4. As I look back on my life all I can see is a lot of failures.
5. I feel I am a complete failure as a person.
- D. 1. I am not particularly dissatisfied.
2. I feel bored most of the time.
3. I don't enjoy things the way I used to.
4. I don't get satisfaction out of anything any more.
5. I am dissatisfied with everything.
- E. 1. I don't feel particularly guilty.
2. I feel bad or unworthy a good part of the time.
3. I feel quite guilty.
4. I feel bad or unworthy practically all the time now.
5. I feel as though I am very bad or worthless.
- F. 1. I don't feel I am being punished.
2. I have a feeling that something bad may happen to me.
3. I feel I am being punished or will be punished.
4. I feel I deserve to be punished.
5. I want to be punished.
- G. 1. I don't feel disappointed in myself.
2. I am disappointed in myself.
3. I don't like myself.
4. I am disgusted with myself.
5. I hate myself.
- H. 1. I don't feel I am any worse than anybody else.
2. I am very critical of myself for my weaknesses or mistakes.
3. I blame myself for everything that goes wrong.
4. I feel I have many bad faults.

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- I. 1. I don't have any thoughts of harming myself.
2. I have thoughts of harming myself but I would not carry them out.
3. I feel I would be better off dead.
4. I have definite plans about committing suicide.
5. I feel my family would be better off if I were dead.
6. I would kill myself if I could.
- J. 1. I don't cry any more than usual.
2. I cry more now than I used to.
3. I cry all the time now. I can't stop it.
4. I used to be able to cry but now I can't cry at all even though I want to.
- K. 1. I am no more irritated now than I ever am.
2. I get annoyed or irritated more easily than I used to.
3. I feel irritated all the time.
4. I don't get irritated at all at the things that used to irritate me.
- L. 1. I have not lost interest in other people.
2. I am less interested in other people now than I used to be.
3. I have lost most of my interest in other people and have little feeling for them.
4. I have lost all my interest in other people and don't care about them at all.
- M. 1. I make decisions about as well as ever.
2. I am less sure of myself now and try to put off making decisions.
3. I can't make decisions any more without help.
4. I can't make any decisions at all any more.
- N. 1. I don't feel I look any worse than I used to.
2. I am worried that I am looking old or unattractive.
3. I feel that there are permanent changes in my appearance and they make me look unattractive.
4. I feel that I am ugly or repulsive looking.
- O. 1. I can work about as well as before.
2. It takes extra effort to get started at doing something.
3. I don't work as well as I used to.
4. I have to push myself very hard to do anything.
5. I can't do any work at all.
- P. 1. I can sleep as well as usual.
2. I wake up more tired in the morning than I used to.
3. I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
4. I wake up early every day and can't get more than 5 hours sleep.
- Q. 1. I don't get any more tired than usual.
2. I get tired more easily than I used to.
3. I get tired from doing anything.
4. I get too tired to do anything.

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- R. 1. My appetite is no worse than usual.
2. My appetite is not as good as it used to be.
3. My appetite is much worse now.
4. I have no appetite at all any more.
- S. 1. I haven't lost much weight, if any, lately.
2. I have lost more than 5 pounds.
3. I have lost more than 10 pounds.
4. I have lost more than 15 pounds.
- T. 1. I am no more concerned about my health than usual.
2. I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body.
3. I am so concerned with how I feel or what I feel that it's hard to think of much else.
4. I am completely absorbed in what I feel.
- U. 1. I have not noticed any recent change in my interest in sex.
2. I am less interested in sex than I used to be.
3. I am much less interested in sex now.
4. I have lost interest in sex completely.

APPENDIX E
Multiple Affective Adjective Checklist

Name..... Age..... Sex....
Date..... Highest grade completed in school.....

DIRECTIONS: On this sheet you will find words which describe different kinds of moods and feelings. Mark an ☒ in the boxes beside the words which describe how you feel now - today. Some of the words may sound alike, but we want you to check all the words that describe your feelings. Work rapidly.

- 1 ☐ active
- 2 ☐ adventurous
- 3 ☐ affectionate
- 4 ☐ afraid
- 5 ☐ agitated
- 6 ☐ agreeable
- 7 ☐ aggressive
- 8 ☐ alive
- 9 ☐ alone
- 10 ☐ amiable
- 11 ☐ amused
- 12 ☐ angry
- 13 ☐ annoyed
- 14 ☐ awful
- 15 ☐ bashful
- 16 ☐ bitter
- 17 ☐ blue
- 18 ☐ bored
- 19 ☐ calm
- 20 ☐ cautious
- 21 ☐ cheerful
- 22 ☐ clean
- 23 ☐ complaining
- 24 ☐ contented
- 25 ☐ contrary
- 26 ☐ cool
- 27 ☐ cooperative
- 28 ☐ critical
- 29 ☐ cross
- 30 ☐ cruel
- 31 ☐ daring
- 32 ☐ desperate
- 33 ☐ destroyed
- 34 ☐ devoted
- 35 ☐ disagreeable
- 36 ☐ discontented
- 37 ☐ discouraged
- 38 ☐ disgusted
- 39 ☐ displeased
- 40 ☐ energetic
- 41 ☐ enraged
- 42 ☐ enthusiastic
- 43 ☐ fearful
- 44 ☐ fine
- 45 ☐ fit
- 46 ☐ forlorn
- 47 ☐ frank
- 48 ☐ free
- 49 ☐ friendly
- 50 ☐ frightened
- 51 ☐ furious
- 52 ☐ gay
- 53 ☐ gentle
- 54 ☐ glad
- 55 ☐ gloomy
- 56 ☐ good
- 57 ☐ good-natured
- 58 ☐ grim
- 59 ☐ happy
- 60 ☐ healthy
- 61 ☐ hopeless
- 62 ☐ hostile
- 63 ☐ impatient
- 64 ☐ incensed
- 65 ☐ indignant
- 66 ☐ inspired
- 67 ☐ interested
- 68 ☐ irritated
- 69 ☐ jealous
- 70 ☐ joyful
- 71 ☐ kindly
- 72 ☐ lonely
- 73 ☐ lost
- 74 ☐ loving
- 75 ☐ low
- 76 ☐ lucky
- 77 ☐ mad
- 78 ☐ mean
- 79 ☐ meek
- 80 ☐ merry
- 81 ☐ mild
- 82 ☐ miserable
- 83 ☐ nervous
- 84 ☐ obliging
- 85 ☐ offended
- 86 ☐ outraged
- 87 ☐ panicky
- 88 ☐ patient
- 89 ☐ peaceful
- 90 ☐ pleased
- 91 ☐ pleasant
- 92 ☐ polite
- 93 ☐ powerful
- 94 ☐ quiet
- 95 ☐ reckless
- 96 ☐ rejected
- 97 ☐ rough
- 98 ☐ sad
- 99 ☐ safe
- 100 ☐ satisfied
- 101 ☐ secure
- 102 ☐ shaky
- 103 ☐ shy
- 104 ☐ soothed
- 105 ☐ steady
- 106 ☐ stubborn
- 107 ☐ stormy
- 108 ☐ strong
- 109 ☐ suffering
- 110 ☐ sullen
- 111 ☐ sunk
- 112 ☐ sympathetic
- 113 ☐ tame
- 114 ☐ tender
- 115 ☐ tense
- 116 ☐ terrible
- 117 ☐ terrified
- 118 ☐ thoughtful
- 119 ☐ timid
- 120 ☐ tormented
- 121 ☐ understanding
- 122 ☐ unhappy
- 123 ☐ unsociable
- 124 ☐ upset
- 125 ☐ vexed
- 126 ☐ warm
- 127 ☐ whole
- 128 ☐ wild
- 129 ☐ willful
- 130 ☐ wilted
- 131 ☐ worrying
- 132 ☐ ...

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APPENDIX F
LEVENSON'S SCALE

Place a check on the line after each statement.

1. Whether or not I get to be a leader depends mostly on my ability.
disagree 1 2 3 4 5 6 agree
2. To a great extent my life is controlled by accidental happenings.
disagree 1 2 3 4 5 6 agree
3. I feel like what happens in my life is mostly determined by powerful people.
disagree 1 2 3 4 5 6 agree
4. Whether or not I get into a car accident depends mostly on how good a driver I am.
disagree 1 2 3 4 5 6 agree
5. When I make plans, I am almost certain to make them work.
disagree 1 2 3 4 5 6 agree
6. Often there is no chance of protecting my personal interest from bad luck happenings.
disagree 1 2 3 4 5 6 agree
7. When I get what I want, it's usually because I'm lucky.
disagree 1 2 3 4 5 6 agree
8. Although I might have good ability, I will not be given leadership responsibility without appealing to those in positions of power.
disagree 1 2 3 4 5 6 agree
9. How many friends I have depends on how nice a person I am.
disagree 1 2 3 4 5 6 agree
10. I have often found that what is going to happen will happen.
disagree 1 2 3 4 5 6 agree
11. My life is chiefly controlled by powerful others.
disagree 1 2 3 4 5 6 agree
12. Whether or not I get into a car accident is mostly a matter of luck.
disagree 1 2 3 4 5 6 agree

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13. People like myself have very little chance of protecting our personal interests when they conflict with those of strong pressure groups.
disagree 1 2 3 4 5 6 agree
14. It's not always wise for me to plan too far ahead because many things turn out to be a matter of good or bad fortune.
disagree 1 2 3 4 5 6 agree
15. Getting what I want requires pleasing those people above me.
disagree 1 2 3 4 5 6 agree
16. Whether or not I get to be a leader depends on whether I'm lucky enough to be in the right place, at the right time.
disagree 1 2 3 4 5 6 agree
17. If important people were to decide they didn't like me, I probably wouldn't make many friends.
disagree 1 2 3 4 5 6 agree
18. I can pretty much determine what will happen in my life.
disagree 1 2 3 4 5 6 agree
19. I am usually able to protect my personal interests.
disagree 1 2 3 4 5 6 agree
20. Whether or not I get into a car accident depends mostly on the other driver.
disagree 1 2 3 4 5 6 agree
21. When I get what I want, it's usually because I worked hard for it.
disagree 1 2 3 4 5 6 agree
22. In order to have my plans work, I make sure that they fit in with the desires of people who have power over me.
disagree 1 2 3 4 5 6 agree
23. My life is determined by my own actions.
disagree 1 2 3 4 5 6 agree
24. It's chiefly a matter of fate whether or not I have a few friends or many friends.
disagree 1 2 3 4 5 6 agree

APPENDIX G

Subject No. _____

Emotion InventoryInstructions:

1. During the recording session, you were asked to view different scenes and to imagine yourself in two situations. We would like you to report the emotion you experienced during each of these conditions by circling the appropriate emotion.

Scene Involving
Reaction Towards
Victim

Anger
Guilt
Sorrow
Fear
Disgust
Happiness
Relief
Grief

RATING _____

Scene Involving
Reactions Towards
Negligent Driver

Anger
Guilt
Sorrow
Fear
Disgust
Happiness
Relief
Grief

RATING _____

2. Please rate your experience during the scenes on a scale from 1 to 10, where "1" reflects very little distress and "10" indicates very strong feelings of distress.

APPENDIX H

Research Subject's Consent Form

Mr. Pratt and his associates are conducting a study to determine the psychological and physiological characteristics of individuals with headaches. If you agree to participate in this study, the following requirements will be expected of you.

First, you will be asked to fill out several questionnaires designed to assess the amount of stress in your life. You will then be connected to a chart-writing polygraph in order to record several physiological measures to include respiration, skin conductance, heart rate, muscle tension of the neck and face, and blood flow to the head.

During the 45-minute session that follows, we will be recording your physiological responses to various situations. In this period, you will be asked to engage in a number of exercises to include mental problem-solving, viewing emotional arousing slides, imagery instructions, and relaxation. We are interested in investigating the physiological changes that accompany different emotional and mental states and have selected this procedure to test various predictions.

Upon completion of the assessment session, you will be disconnected from the polygraph and will receive an explanation of the objectives of the present study. At this time, we will attempt to answer any questions you may have concerning the experiment.

It is important that you are aware of the following conditions:

- 1.) Any information you provide, from interview, testing and the physiological assessment will be held in confidence.
- 2.) You have the right to terminate your participation in this study at any time

If the terms of the present study are agreeable to you, please indicate this by providing your signature below.

Signed: _____

Date: _____

APPENDIX I

Mental Test

1. What is the capital of Italy?
2. What is the population of the United States?
3. What color results from mixing red and yellow?
4. What is the next number in the following series? 1, 2, 4, 7, 11, ____
5. Which country recently elected a socialist leader?
6. How many inches are there in 100 cm?
7. Big is to small as summer is to ____?
8. How many quarters are there in \$8.75?
9. What mixture of colors results in purple?
10. Which word does not fit in with the others?
Liquidate, eradicate, evaporate, annihilate
11. What is the distance between New Orleans and Baton Rouge?
12. The country of Equador is found in which continent?
13. Who were the two U.S. presidential candidates in the 1960 election?
14. What is the square root of 121?
15. What is the largest state in the United States?
16. Scarlett O'Hara is the leading character of what famous American novel?
17. Seventy percent of 500 is equal to?
18. What do the letters in OPEC stand for?
19. Who is credited with formulating the law of gravity?
20. In what year did American Astronauts first set foot on the moon?

APPENDIX J

Table A

Paired t-Tests for Fear-Anger Comparisons

Variable	df	Means		t-value	p
		Fear	Anger		
Frontalis EMG	29	1.36	1.06	.99	.332
Trapezius EMG	29	1.95	1.47	.84	.407
Temporal Artery Pulse Volume	29	4.97	5.21	-.44	.665
Respiration Frequency	29	-.77	-.85	.15	.879
Respiration Amplitude	29	19.83	17.42	.74	.464
Skin Resistance Level	29	-22.36	-25.95	.85	.401
Skin Conductance Response	29	-.01	-.02	-.52	.609
Heart Rate	29	4.80	4.20	.56	.578

Appendix K

Table B

Group Frequencies on Emotion Survey Validity Check

Fear Condition

EMOTION	Group			TOTAL
	MIG	MCH	CON	
Anger	1	2	2	5
Guilt	0	0	0	0
Sorrow	6	2	4	12
Fear	2	3	1	6
Disgust	1	0	0	1
Happiness	0	0	0	0
Grief	0	3	3	6
Relief	0	0	0	0
				<u>30</u>

Anger Condition

EMOTION	Group			TOTAL
	MIG	MCH	CON	
Anger	5	2	2	9
Guilt	0	0	0	0
Sorrow	3	3	4	10
Fear	0	2	0	2
Disgust	2	2	1	5
Happiness	0	0	0	0
Grief	0	1	3	4
Relief	0	0	0	0
				<u>30</u>

APPENDIX L

Table C

Pearson Correlation Values for Psychophysiological Relationships

Self-Report Measures	Selected Physiological Variables								
	Mental Test			Fear			Anger		
	Front EMG	Trap EMG	TPV	Front EMG	Trap EMG	TPV	Front EMG	Trap EMG	TPV
Beck Depression Inventory	-.091	-.090	.156	.388*	-.070	-.076	.197	.009	-.055
MAACL									
Anxiety	-.027	-.371*	.098	-.146	.218	-.160	-.069	.308*	-.288
Depression	-.092	-.352*	.196	-.093	.302	-.173	-.032	.304*	-.208
Hostility	-.207	-.348*	.055	.018	-.031	-.139	.118	.066	-.237
Life Stress									
Quantity	.351*	.401*	.102	-.112	.036	-.229	.004	.074	-.140
Recency	.106	.152	.099	-.041	-.151	.013	-.056	-.182	.091
Subjective	.149	.058	.098	.132	.096	.101	-.437	.117	.115
Levenson's Scale									
Internal	.166	.262	.163	-.172	-.023	.339*	-.317*	-.146	.338*
External	-.199	-.308	-.138	.179	.140	.119	.121	.116	.175
Powerful Others	-.411*	-.182	-.188	.190	.156	.108	.159	.121	.046
SUDS Scales									
Fear	.106	-.237	.139	.256	-.044	-.139	.237	-.079	-.006
Anger	.067	-.255	.019	.318*	.036	.000	.360*	-.014	.113

Front = Frontalis; Trap = Trapezius; TPV = Temporal Pulse Volume.

* $p < .05$.

VITA

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EDUCATION:

1968-1969 Southern Illinois University Carbondale IL
Major: Psychology Minor: Design
B.S., June, 1974

1974-1976 Tulane University New Orleans LA
Major: Personality
M.S., December, 1976

1978-1982 Louisiana State University Baton Rouge LA
Major: Clinical Psychology Minor: Behavioral Neurology
Ph.D., August, 1982

CLINICAL EXPERIENCE:

1981-Present Clinical Intern Palo Alto VAMS Palo Alto CA
Description of Training: Treatment of veterans and their families in four settings-
(1) Family Program. Emphsizing systems approaches to family therapy.
(2) Health Psychology. Group and individual treatment involving psychotherapy, stress management, biofeedback, relaxation, and pain control procedures.
(3) Satori. Inpatient therapeutic community for treatment of drug and alcohol abusers.
(4) Mental Hygiene Clinic. Outpatient case management.

1981 Consultant Social Science Consultants New Orleans LA
Description of Duties: Industrial consulting responsibilities to include personnel selection, program development, and organizational analysis.

1980-1981	<p>Practicum Student New Orleans VAMC New Orleans LA</p> <p>Description of Training: Assessment and treatment of veterans and their families, and in-service training, through three clinics-</p> <p>(1) Inpatient Psychiatric Ward, Assessment and individual and group psychotherapy.</p> <p>(2) Neuropsychology Unit. Assessment and consultant services.</p> <p>(3) Drug and Alcohol Ward. Inpatient milieu therapy approaches.</p>
1980	<p>Psychology Extern Earl K. Long Hospital Baton Rouge</p> <p>LA Description of Duties: Assessment, treatment, and consultation in outpatient family practice and inpatient pediatric ward settings. Also, in-service training for nurses and medical residents.</p>
1979-1981	<p>Psychological Assistant Baton Rouge Hospital Baton Rouge LA</p> <p>Description of Duties: Private practice-connected psychological assessment of adults and adolescents in drug and alcohol programs.</p>
1979-1980	<p>Practicum Student LSU Student Health Baton Rouge LA</p> <p>Description of Training: Evaluation and individual and group treatment of college students in a mental health clinic.</p>
1977-1978	<p>Practicum Student Southeast LA State Hospital</p> <p>Mandeville LA Description of Training: Psychological assessment of recent admissions into adult and adolescent inpatient wards.</p>
1976-1977	<p>Psychology Assistant Regina Coeli Child Center</p> <p>Covington LA Description of Training: Consulting for rural Head Start Program involving child assessment and treatment, parent and teacher training, and program development.</p>
1976-1978	<p>Researcher LSU Medical School New Orleans LA</p> <p>Description of Duties: Technical responsibilities for Heart, Lung, and Blood Institute-funded study of blood pressure control factors.</p>
1974-1981	<p>Testing Supervisor Tulane University New Orleans LA</p> <p>Description of Duties: Supervision and administration of national exams for Educational Testing Service, Psychological Corporation, and other certification programs.</p>

1972-1974 Administrator Carbondale Free Clinic Carbondale IL
Description of Duties: Administrative and paramedical responsibilities in a community-supported medical and counseling clinic. Group therapy for drug abuse program also provided.

MILITARY EXPERIENCE:

1970-1972 Medical Specialist U. S. Army Medical Corps Berlin, Germany
Description of Duties: Service rotations included emergency medicine, Honorable Discharge, 1976.

TEACHING EXPERIENCE:

1980-1981 Teaching Assistant LSU Psychology Department Baton Rouge LA
Description of Duties: Instruction, coordination, and supervision of students in advanced projective course.

1979-1980 Teaching Assistant LSU Psychology Department Baton Rouge LA
Description of Duties: Instruction, coordination, and supervision of students placed in community psychology practicum sites.

1978-1979 Instructor Ecole Classique New Orleans LA
Description of Duties: Instructor for two high school-level psychology courses.

1979-1980 Instructor Meadows-Draughton Business College New Orleans LA
Description of Duties: Instructor for courses in management and industrial psychology.

PUBLICATIONS:

Tuma, J. M., & Pratt, J. M. Clinical child practice and training: A survey. Journal of Clinical Child Psychology, 1982, 11, 27-34.

Williamson, D. A., Pratt, J. M., Cohen, R. A., & Monguilliot, J. Controlled outcome evaluation of self-help and therapist-assisted treatments of muscle contraction and migraine headaches. Under editorial review.

PRESENTATIONS:

"Self-recognition and self-esteem: An analysis of perceptual, physiological and self-report indices." Southeastern Psychological Association, Hollywood, Florida, May 1977. (with Fryear)

"Psychological differences among headache sufferers." Association for the Advancement of Behavior Therapy, Toronto, Canada, November, 1981. (with Williamson et al.)

"An investigation of empirically-derived categories of headache." Association for the Advancement of Behavior Therapy, Toronto, Canada, November 1981. (with Williamson et al.)

"Controlled evaluation of a self-help relaxation program for the treatment of headache." Association for the Advancement of Behavior Therapy, Toronto, Canada, November 1981. (with Williamson et al.)

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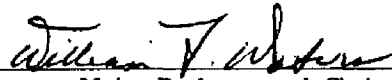
EXAMINATION AND THESIS REPORT

Candidate: J. Mark Pratt

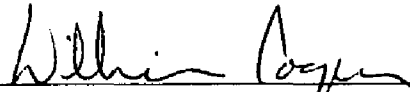
Major Field: Clinical Psychology

Title of Thesis: Headache Sufferers' Psychological and Physiological Reactions to Stress

Approved:

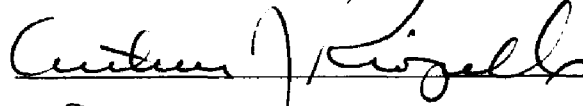


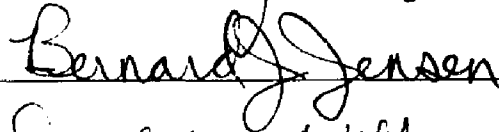
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Date of Examination:

September 13, 1982